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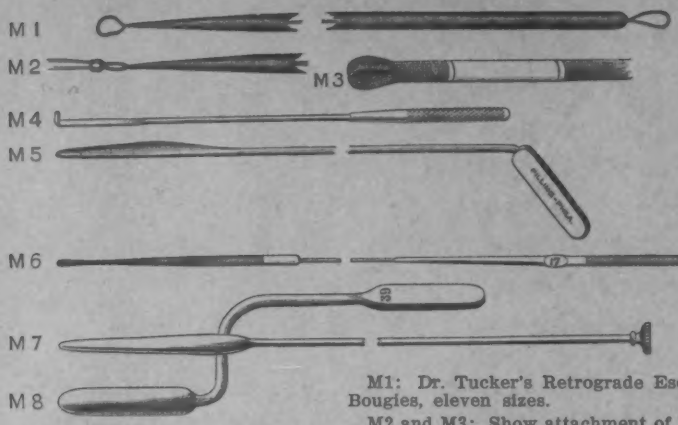

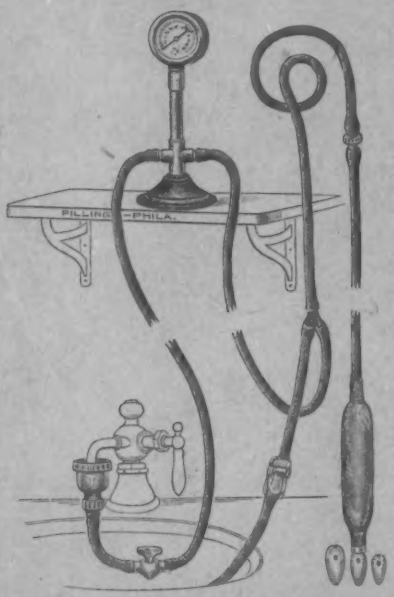

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ORIGINAL COMMUNICATIONS.

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SYMPOSIUM ON HEADACHE.

HEADACHES FROM THE STANDPOINT OF THE OPHTHALMOLOGIST.*

DR. JOHN GREEN, St. Louis.

With the wide dissemination among the laity of medical knowledge (some of it grossly perverted, to be sure), it has become customary for many sufferers from headache to make their own diagnosis of the origin of this symptom and to consult, primarily, that specialist in whose field lies the assumed cause. Thus we see patients with hyperopic astigmatism consulting the internist, others with headache associated with hypothyroidism visiting the rhinologist, those with impacted third molars and frontal sinusitis seeking help from the ophthalmologist, and so on.

What a splendid thing it would be and what a saving of lost motion if all headachy patients could first be passed through a diagnostic clearing house, there to be surveyed and finally allocated to the man who could really do them some good! Possibly this ideal has been attained in some great clinics. Certainly, it has not even been approximated in St. Louis.

It is easy, indeed, to accept the situation as presented to us by the patient and to encourage him in the belief that he has chosen his doctor wisely and that relief is within our power to give. But supposing we do our utmost and then find that the patient is wholly

*Read as part of a Symposium on Headache, before St. Louis Ear, Nose and Throat Club, Dec. 21, 1927.

unrelieved? Not only is such an outcome unfair to the patient, but tends to bring discredit on the profession. If we find it impractical to secure for our patients a preliminary diagnostic survey, how may we hope to avoid the mistake of applying a remedy to a disorder or malfunction which in reality does not exist? In an attempt to solve this problem I have found it helpful to start with the assumption that, in all probability, the cause of the headache lies outside of my own field and, while neglecting nothing in the way of ophthalmic diagnosis and therapy, try, in the end, to be in a position intelligently to refer the patient, or be forced to acknowledge that, after all, the cause lies in the domain of ophthalmology.

It is of utmost importance to secure an accurate picture of the circumstances which give rise to headache. I cannot stress too strongly the value of an adequate history of the headache, not only in the patient, but in the patient's family as well. Lancaster has well said, "The typical eye headache is rarely an isolated phenomenon in a family of any size".

The character and location of the pain is not pathognomonic of an eye headache; probably most headaches due to the focusing mechanism (error of refraction) are frontal, while those due to anomalies of the adjusting mechanism (muscle imbalance) are more likely to be vertical or occipital.

Ocular headaches may be divided into *a.* those depending on some error of refraction or muscular anomaly, and *b.* those connected with or dependent on a true ocular disease (iritis, glaucoma, keratitis, etc.).

In considering the first group, it should be borne in mind that only a very small percentage of those who are wearing glasses have been accurately fitted. Most individuals secure their glasses primarily from the refracting optician (optometrist), whose methods are inadequate to disclose the true refractive state of the eye. It is the oculist's daily experience to encounter patients wearing glasses utterly at variance with the true refractive state. Many a person with headache solely dependent on refractive error secures these improper glasses. As the headache is unrelieved or possibly made worse, he naturally assumes that the headache is not of ocular origin and seeks relief from another source, often from a representative of one of the cults.

Our inquiry should determine the duration of the headache, its occurrence in connection with any kind of use of the eyes, its association with ocular pain or discomfort, the simultaneous occurrence

of blurring or swimming of the letters and the existence of marginal blepharitis and conjunctivitis.

Sometimes an evening at the movies is followed by a headache the following morning. There are patients who are not in the least incommoded during or after viewing a well-photographed film drama, but who experience ocular fatigue and browache after looking for a few minutes at the animated cartoon. In the interest of ocular welfare, I wish that this form of movie might be abolished. The effect is gained by presentation in rapid succession of photographs of slightly dissimilar attitudes of animals or objects drawn by the cartoonist. There is no even flow from one picture to the next; the effect is jerky and a travesty of true motion, and is very fatiguing to the retina. I fear that this hope will never be realized so long as these cartoons are so highly appreciated by the younger patrons.

The farsighted and astigmatic eye uses its adjusting mechanism to compensate for the refractive defect. If the patient is youthful and in good general condition, it is surprising how much farsightedness can be carried without symptoms. Let there come, however, a period of excessive work, as in preparing for examinations, or a spell of sickness, and the adjusting mechanism is no longer able to cope with the situation, and headache results. Then it is that glasses become necessary to tide the patient over the period of ciliary muscle weakness. When the general bodily tone is restored the adjusting mechanism regains its ability to cope with the situation and glasses may be discarded.

Many individuals can carry without symptoms a moderate degree of farsightedness or farsighted astigmatism up to the age of 40 or thereabouts when, all of a sudden, the approach of presbyopia throws an unwonted strain on the hitherto perfectly functioning eye and there is a rather sudden collapse.

The ability to refract the human eye accurately is an art that is only acquired after long practice. Several factors militate against accuracy. First, the hyperactivity or unequal activity of the ciliary muscle. Second, the unresponsiveness or lack of intelligent co-operation on the part of the patient. We can subdue the ciliary muscle by various methods, such as fogging or its variants, or the use of an efficient cycloplegic. The ability to extract the truth or an approximation of the truth from the patient who has never in his life observed anything accurately, is a task that calls for all the resourcefulness of the examiner. The job is a slow, painstaking and often nerve racking affair for both oculist and patient. There are many

patients who dread the refraction test much more than they do the manipulations of the dentist.

The eye is a living organism and as such is subject to change. This change may be extremely minute, but it is these minute changes that affect the curvature of the cornea, the elasticity of the lens, the tonus of the ciliary muscle, and the position of the percipient element, the retina. A change in the anteroposterior diameter of the eye of not more than one-hundredth of an inch will suffice to blur the image.

Hardly less important than errors of refraction are certain types of muscular imbalance in the production of ocular headaches. Most troublesome and provocative of great annoyance and inability to use the eyes is insufficiency of convergence. With these patients all goes swimmingly so long as they do not attempt any near work. When any near task is indulged in there is almost immediate distress—eyeache and browache, which soon compels abandonment of the task.

Oculists encounter the following type of cases with a fair degree of frequency. The patient comes complainng of ocular and retroocular headache of a week or more duration. This is accompanied by inability to use the eyes for any length of time. Inspection reveals a moderate conjunctival congestion, a slight droop of the upper lids and, what is most characteristic, a little edema of the lower lids. Vision is unaffected; nerve heads may be slightly congested. Such a picture means to me an acute frontal or an anterior ethmoidal sinusitis and my presumptive diagnosis has almost always been confirmed by the consulting rhinologist. It seems likely, therefore, that the chronic affections of the frontal and ethmoidal cells may have a like influence, though in lesser degree.

The desire for binocular single vision is pre-eminent and it is the task of the individual to secure this vision if at all possible. This necessitates, in cases of improper muscular insertion, the transmission of unduly strong nervous impulses to certain muscles in order that diplopia may be avoided. We are hampered in the study of the function of the ocular muscles by this desire for single vision which tends to mask the true state of the muscles. Atropine will temporarily paralyze the ciliary muscles, but has no effect on the extrinsic ocular muscles. In order to secure a fairly accurate notion of the behavior of the ocular muscles of one eye, we must dissociate that eye from the influence of its fellow. This can be effected by covering one eye for a period of from several hours to several days. In this manner both eyes are permitted to come to a state of muscular rest. At the end of such a prolonged occlusion one is often sur-

prised to find how greatly at variance are the measurements of the imbalance as compared with the initial measurements. In such cases the complete correction of the vertical defect, hyperphoria, and the partial correction of the lateral defect is often marvelously efficacious in securing relief from headache.

Many a patient with marked retinal angiosclerosis is also a victim of headache which more than likely is a part of the clinical picture of a generalized vascular hypertension and should be referred to the internist for general medical study. Nor need I dwell on the futility of the ophthalmologist attempting to control a headache accompanying a choked disc without consultation with a neurologist or neurological surgeon.

Of the ocular diseases closely associated with headache, the two most conspicuous are iritis and chronic glaucoma. The former inevitably comes under the care of an oculist early in its course by reason of the acute inflammatory appearances and pain. With glaucoma, however, it is a different and sadder story. The dull ache, frontal and temporal, does not always seem to the patient to originate in the eyes. He may or may not have colored halos. He often does not lose much central vision until the disease is far advanced. His early appeal is to the family physician, who only rarely suspects anything wrong with the eyes. Here let me state without equivocation that an elderly patient with persistent headache that does not yield to ordinary measures, should have a thorough ocular examination. In this way only will be averted many eye tragedies, for indeed glaucoma has well been called the tragedy of ophthalmology. Controllable in its earlier stages, it leads, when not treated, inevitably to blindness.

If we are ever to lessen blindness from this source the whole profession must awaken to the possibility that persistent dull headache in elderly persons may and often does have glaucoma as its basis.

There are many cases in which there may be a reasonable doubt as to whether the headache is ocular or not. In such, various tests may help: Atropinization over a period of a week will stop any headache due to hypertonicity of the ciliary muscle. Covering both eyes for a similar period will have the same effect. The simple and harmless therapeutic test of wearing accurately prescribed spectacles will lead to some conclusion. If the headache is due to overstrain of the fixing mechanism in the interest of binocular vision, prolonged occlusion of one eye will settle the matter and at the same time give accurate indications for the incorporation of prisms in the glasses.

A word should be said of certain retinal conditions, sometimes congenital, which impair vision but do not destroy it. Victims of such anomalies have an unfortunate predilection for jobs requiring the finest adjustments, *e. g.*, bookkeeping, draughtsmanship, watchmaking, etc. With the best correction obtainable their vision may be less than one-half and the attempt to carry out the fine tasks incident to their work is often accompanied by severe headache. In order to recognize the print, or line, or figure, the object looked at is held very close, enlarging the image to be sure, but throwing a fearful strain on the muscle of accommodation.

Thanks to the efforts of illuminating engineers, many of the problems of illumination in relation to eye use have been successfully solved. But, unfortunately, this knowledge is not universally applied, and some ocular headaches (*i. e.*, headaches due to the use of the eyes) are not dependent on any structural defect in the eyes or muscular imbalance, but are solely dependent on faulty or insufficient illumination. I may cite as a conspicuous example of wretched illumination the auditorium of the St. Louis Medical Society.

The management of some headachy patients may be simplicity itself, in others the origin may be utterly baffling and will tax the therapeutic acumen of the collective faculty, but none of us should lie down on the job and all should appreciate the value, nay, the necessity of effective teamwork with our colleagues in other branches of medicine.

1002 Beaumont Building.

HEADACHES: THEIR NEUROSURGICAL ASPECT.*

DR. ROLAND M. KLEMME,† St. Louis.

The subject assigned to me in this symposium is headache due to neurosurgical conditions. Headache is such a common complaint and falls into the field of so many specialists that a complete discussion would infringe on almost every special field, therefore I shall limit my remarks strictly to headaches due to neurosurgical conditions.

There is no symptom of which patients complain more than headache. Frequently patients use the term "headache" to describe a pain in the face or scalp so that it may be very difficult for a physician to determine exactly what the patient means. The patient may use this term to describe deep-seated pain in the head, or localized pain in the head or scalp, or even pain that radiates over the head and face following the course of peripheral nerves.

There is a tendency of the specialist to find a cause for this symptom in his own special field, frequently forgetting to consider the patient as a whole. In order to avoid slipping into such an error, the most important guide we have is a carefully taken history and a complete physical examination. Unless this is done the real cause of the headache may frequently be missed and the patient may be subjected to unnecessary forms of treatment that will have little or no effect.

Operations under local anesthesia lead one to believe that the structure in the cranial cavity which is particularly sensitive to pain is the dura, while the pia arachnoid and the brain itself seem to be insensitive to ordinary pain stimuli. Pressure or dragging on sensory or mixed nerves in the intracranial cavity also produces pain.

Headaches may be classified in many ways, but from a neurosurgical standpoint it seems most useful to differentiate those of intracranial origin from those of extracranial origin. This is the classification that I shall make use of here. The principal types of headaches may be grouped under the following headings:

Intracranial: 1. Increased pressure: *a.* New growths: primary; metastatic; tuberculoma and gumma. *b.* Trauma: focal; general. *c.* Infections: abscess; meningitis. *d.* Aneurysms.

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*Read before the Otolaryngological Club of St. Louis.

†From the Department of Surgery, Washington University School of Medicine, St. Louis.

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Extracranial pain, but confined to head and face, of which neuralgias are most frequent. *1.* Cranial nerve neuralgias: trigeminal neuralgia; Gasserian ganglion tumors; glossopharyngeal neuralgia; occipital neuralgia; postauricular neuralgia and others. *2.* Cranial nerve neuritis.

In addition to the conditions above mentioned, which all may be responsible for headache, everyone encounters cases of headache in which no assignable cause can be found at all. There is, however, one outstanding fact which has been forcibly impressed on the neurosurgeon, namely, that the dura, just like the parietal peritoneum and parietal pleura, is particularly sensitive. When the dura is subjected either to stretching or inflammation or some other form of distortion, this is quite apparent. The fact that there are certain points in the skull where the dura is particularly firmly adherent may be an important factor in headache and, due to these points of attachment, any alteration in pressure or increase in brain bulk readily puts the dura under tension. Sudden increases of tension, as are seen in increased intracranial pressure, commonly produce headaches. Whether this is the explanation for so-called "spinal puncture headache" is not quite so clear. The withdrawal of fluid in these cases sometimes is followed by headaches and it may be that the withdrawal of the cerebrospinal fluid causes the cerebral hemisphere to drag on the dura. On the other hand, it is usually found that so-called "lumbar puncture headache" only develops several hours after the puncture and not immediately after the withdrawal of fluid. This may be due to the increased production of cerebrospinal fluid which is a well known phenomenon after cerebrospinal fluid has been removed.

No symptom is more commonly seen with intracranial new growths than headache. This is unquestionably due to the increased intracranial pressure and tension on the closed dural envelope. This is readily proven by the striking relief that patients experience after the dura has been widely opened from a properly placed decompression operation. It is, however, striking that there is great variability in the severity of the headache and while some brain tumors are accompanied by terrific headache while still very small, others attain a great size and are accompanied by little headache. Unquestionably the location of the tumor, as well as its rate of growth, have much to do with this, more than the actual size of a tumor. Thus a very small tumor in the posterior fossa obstructing the aqueduct of Sylvius by producing an internal hydrocephalus may raise the intracranial pressure so rapidly that the patient suffers intense headaches.

A slow growing tumor presumably accommodates itself and the surrounding brain tissue so that there is little or no headache. This seems the only reasonable explanation of why patients with huge tumors may not complain of headache. Whether the actual involvement of the dura by a tumor plays a role in producing headache is not clear. The intense headaches that patients with metatastic tumors complain of is probably explained by the rapid rate with which metastases grow.

Whether another factor is concerned in the production of headache in the case of tuberculomas and gummas is an interesting problem about which to speculate, for here there is an inflammatory disease and it may be that the inflammation plays a role in the production of headache in addition to the actual tumor. The infratentorial lesions, of course, may cause an early blocking of the cerebrospinal fluid at the aqueduct of Sylvius just as in any other type of tumor and consequently these patients are more likely to have headaches early in their disease than patients with supratentorial lesions.

One of the most outstanding symptoms encountered in patients suffering with skull injury is headache. This may appear immediately after the injury and may continue for a long time, even years, or it may come on immediately after the injury and disappear in the course of a few days or weeks, and then again it may only appear some weeks after the injury and be either transient or persistent. Just why the headache in patients with skull injuries should assume these different forms is not readily explained. There are numerous factors that may be responsible. In the first place, the trauma to the tissues, bone, periosteum or dura may be responsible; in the second place, it may be due to the increased intracranial pressure, which is a very constant accompaniment of head injuries; and not infrequently, we have learned in the last few years, the cause of this increased intracranial pressure is the edema of the brain which accompanies any contusion or laceration no matter how slight. These headaches may be generalized or only in a particular region of the head. When they are generalized, the increased pressure is probably responsible for them, while localized pain is more likely to be due to trauma of the soft tissues or possibly to the injury to the periosteum seen in a depressed fracture.

What the factor is in headache in cases of middle meningeal hemorrhage or torn arachnoid is not perfectly clear. Presumably it is the increased intracranial pressure for when the pressure is relieved the headache very promptly subsides.

The headaches occurring in localized infections of the brain abscesses and meningitis, probably have two underlying causes, one the inflammatory process, and the other, the increased intracranial pressure due to the lesion. How much each of these factors is responsible it is very difficult to determine, for many cases of meningitis are seen complaining of intense headaches that, as far as any tests indicate, have no appreciable increased intracranial pressure. In such cases one is forced to conclude that the inflammatory process is the causative factor.

Just what the cause of headache is in aneurysm of the circle of Willis or its branches presents still another interesting problem. For here, in addition to the increased intracranial pressure, the aneurysm may be causing direct pressure on a nerve. Since aneurysms of the internal carotid lie very close to the Gasserian ganglion, it is not surprising that patients with this condition refer their pain to the distribution of the fifth nerve, and though they call this headache it really is more in the nature of a neuritic pain.

Extracranial Headaches: This term is used since patients so frequently do not or are not able to distinguish between a true headache of intracranial origin and a pain in the face or head. Such pains, however, are better described as neuralgia than true headache. The most familiar example of this sort of neuralgic pain is trigeminal neuralgia, or tic douloureux. This we know is due to some disturbance in the Gasserian ganglion itself, but thus far no histological examinations have revealed any definite pathological changes which adequately explain the intense suffering that these patients experience. The pain in these cases is spasmodic in character and in every way resembles the pain seen in neuritis. The bizarre pains in the face that patients complain of which are variously ascribed to infected sinuses or diseased teeth are often quite indistinguishable from the true neuralgic pains just mentioned. In both types there is no sound underlying pathology of the nerve itself that has ever been described. In recent years the multiplicity of bizarre pains has been still further complicated by the recognition of a glossopharyngeal neuralgia which is due to disease of the ninth nerve, just as tic douloureux is due to disease of the fifth nerve, but there, too, no pathological changes have been described to account for the condition.

Up to the present point, I have outlined in a general way the causes of headaches and pains in the face due to a variety of neurosurgical lesions. I now propose to take up somewhat more in detail some of the salient associated symptoms which characterize these pathological processes. These symptoms, irrespective of their cause,

may be divided into general and focal ones. The most familiar ones, in addition to headache, are vomiting, nausea and loss of vision. The ones less well recognized, but equally important, are double vision when due to paralysis of one sixth nerve, sighing respiration, yawning, picking at the bed clothes and mental dullness. These are due to increased intracranial pressure and the most frequent cause of increased intracranial pressure is some form of brain tumor. In recent years we have seen numerous cases of brain tumor in which many of the cardinal general symptoms were absent but the fact remains that, in the vast majority of cases, at least some of the general symptoms on careful examination will be found. Of these, none is more frequent than choked disc. Consequently, it is not putting it too strongly to say that every patient with headache ought to have, as a matter of routine, an ophthalmoscopic examination. When once this becomes a routine procedure, many a tragedy might be avoided and preventable blindness could be obviated.

A further aid in recognition of these cases of increased intracranial pressure, of course, is the X-ray, which may show very characteristic changes both in adults and children.

The practice of taking manometric readings of the cerebrospinal fluid to determine increased intracranial pressure is not only unsatisfactory but is attended by a great deal of risk. Repeated deaths have resulted from this procedure and therefore it should be resorted to only after weighing the advantages to be derived from such information against the dangers of the procedure.

The focal symptoms in a given lesion are so numerous that in a general discussion of this sort they can hardly be taken up in detail. They vary, depending on the particular anatomical region of the brain that is affected.

The relief of headaches is naturally the ultimate object for which we all strive and in order to do this effectively my preceding remarks have, I trust, made it clear that the underlying cause of the headache must first be determined. When a new growth is responsible, the most effective way to deal with it is to try to remove it by a surgical procedure. Temporary relief may be afforded by the administration of hypertonic fluids intravenously, per rectum, or by mouth. These have only a very transient effect and are used preparatory to operation. The solutions most frequently used intravenously are glucose and sodium chlorid, while for rectal and mouth administration magnesium sulphate is more effective. Narcotics for tumor headache not only are unsatisfactory but are frequently attended by a good deal of risk.

The treatment of headache following trauma offers an even more difficult problem than in the condition just mentioned, for these headaches frequently are continuous and incapacitating. Physical examination of these patients may be entirely negative and there may be no other general or focal signs. In some of these cases the administration of hypertonic fluids over a prolonged period helps. This is best given in the form of cathartics. Mild analgesics and rest in bed might also be of great benefit. When all these fail, decompression operation may relieve the patient. Very recently Penfield has been able to relieve some of these patients by the injection of air into the spinal subarachnoid space but his observations are so new that considerably more work will have to be done before this can become a routine measure. When a depressed fracture is present, the elevation of the fracture may prevent the future devlopment of headache and for this, as well as other reasons that I shall not go into here, all depressed fractures should be operated. The accumulation of cerebrospinal fluid in the subdural space, due to a rupture of the arachnoid, probably occurs very much more frequently than we have been led to believe in the past, and since this, in addition to producing other symptoms, may be a frequent cause of headache, operation may be indicated.

When the headache is neuralgic in origin, on the other hand, treatment must be directed to relieve the underlying cause. The most frequent type that the neurosurgeon is called upon to deal with is the trigeminal neuralgia. Once the diagnosis is definitely established, the treatment is perfectly clear. In mild cases, alcohol injections are temporarily effective. When severe, however, the extraction of the posterior root of the Gasserian ganglion is the only effective method of treatment.

In summing up, therefore, the subject of headache, I would say this: Before instituting any treatment, each and everyone of us ought to exhaust every means to find the underlying cause of the headache and then and only then should treatment be instituted. If the cause of the headache falls in the province of the neurosurgeon, he should deal with it; on the other hand, unless he is certain of this, it is far better for him to leave it to others to determine the cause than to institute inadequate treatment.

519 University Club Building.

HEADACHE FROM THE STANDPOINT OF THE GYNECOLOGIST.*

Dr. George Gellhorn, St. Louis.

Every specialty, it seems, passes through the three stages of discovery, expansion and readjustment. In the first stage a few men here and there discover in the vast acreage of medicine a field heretofore unnoticed, which they begin to till with vigor and aptitude. In the stage of expansion the followers of these pioneers extend the work far beyond its original boundaries. In the final stage, that of readjustment, the workers are reaching the limits of their possessions and perceive, beyond them, other domains, borderlands, and they build bridges across the boundary lines to get into touch with their neighbors.

This last phase is obviously in process in your Society. When specialists begin to criticize themselves, to analyze their own results, to consider their work as a part of Medicine, not as Medicine itself —then they establish their specialty on a firm and enduring basis. To change the metaphor, we specialists, of whatever denomination, have sprung from the same source and if we are true to our calling, we must eventually strive back to our common mother, the art of healing. That this path is beset with obstacles, that we are often led astray by our own glittering achievements, I need not emphasize. Some of us are still too engulfed to find our direction; and it is not so very long since that Abraham Jacobi accused the gynecologists of "looking at the world through a vaginal speculum".

A symposium on headache, this most common symptom encountered in every specialty, is, therefore, bound to be of mutual benefit; and each contributor is certain to receive as much as he gives. My own contribution is largely based on personal observation. For, strange as it may seem, there is no literature on the subject in the realm of gynecology and obstetrics. Sporadic references of a line or two, never more, may be found, but you would seek in vain for the word "headache" or its equivalents in the index of large reference works and textbooks. Yet, almost every other gynecologic patient enumerates this among her complaints, and the gynecologist must have some idea of the classification and causation of headache.

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*Read (by invitation) in a Symposium on Headache before the St. Louis Ear, Nose and Throat Club, on Dec. 21, 1927.

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In presenting the subject to you, I propose to group the cases of headache as I observe them in practice, in two large categories: those in which there is but a loose connection between the headache and the underlying condition, and those in which the headache is directly caused by the condition in question.

Foremost in the first category is what I may briefly designate as *nervous* headache. It is felt chiefly in the occiput from where it extends as a pulling sensation to the back of the neck, and as a pressure on top of the head. Less frequently does it occupy other parts. It is almost invariably associated with other neurasthenic symptoms, such as insomnia or, on the contrary, a very heavy, yet not refreshing sleep; frequent desire to urinate, more often by day than during the night; irritability; hyperesthetic zones of the skin, chiefly of the lower abdomen; palpitations, etc. This syndrome is found in so large a number of widely different gynecological conditions that it cannot possibly be considered specific. Rather must it be explained on the ground of neurasthenia, which is so common in female patients. In these, neurasthenic manifestations may break out under any strain; and often enough the wear and tear of mere living may be the exciting cause. If there happens to be a more or less distressing pelvic lesion, the anxiety and attention which are easily focused on the genital sphere, are responsible for the appearance of this particular form of headache and kindred symptoms. In order not to be misled into construing an imaginary connection between the pelvic condition and the headache, I am in the habit of prescribing bromids and re-examining such patients a few days later. By that time, the headache and nervous phenomena have disappeared, and the remaining symptoms may now safely be regarded as being caused by the existing genital affection.

In patients with bleeding fibroids or other pelvic affections which give rise to protracted hemorrhages, we not infrequently encounter a headache which, as a rule, is located in the temples. This is the *anemic* form of headache and in no wise differs from that observed in any anemic individual. The appearance of the patient, her pale mucous membranes, her quick fatigue on slight exertions, and the low blood pressure render a determination of the hemoglobin almost superfluous, inasmuch as the usual tests for the latter are highly unreliable; if there still be doubt, the red blood count will clinch the diagnosis. Whatever form of therapy the underlying gynecologic condition may require, the coexisting headache calls for treatment of its own. Such treatment consists, in the main, of iron and arsenic and should be combined with cod liver oil and a strengthening dietetic and hygienic regime.

. When the nervous and anemic headaches are associated, as is often the case, a combination of the appropriate treatment must be instituted.

Toxic headaches are rather frequent in gynecologic patients. Their most common cause is constipation, which but rarely has any connection with any pelvic disease that may exist at the same time.

In former days, *reflex* headaches played a prominent role in the symptomatology of gynecological diseases. Retroflexion of the uterus, tumors of the ovary, in fact, every big or slight deviation from normal conditions within the pelvis were held responsible for the headache if that symptom happened to be in evidence. Since we gynecologists have ceased to ascribe any and every ailment to disorders within the genital sphere, the word "reflex" has disappeared from our vocabulary.

The second large category of headaches comprises the instances where this symptom seems to be directly caused by the pelvic condition. We may call these headaches *specific* and conveniently subdivide them as to their occurence in gynecology and obstetrics.

In gynecology, we first have to mention *menstrual* headaches. This refers to individuals who suffer from headaches only at the menstruation but are entirely free from it in the intermenstrual period. In some cases, headache is the only symptom complained of; more often is it associated with other disturbances of health at that time. The women are "unwell". There is a general lassitude, both physical and mental; the gastrointestinal tract is affected; the timbre of the voice suffers; an eruption of acne may appear; above all, a severe headache, unilateral or diffuse, may occur either immediately before or during the flow. While this form of headache has been explained by some solely on the ground of vasomotor changes, the prevailing view now inclines to seek its origin in a sort of ovarian toxemia. This conception is strengthened by the very multiplicity of the general menstrual symptoms, and is reinforced by the fact that all these alterations of the general condition disappear quickly as soon as a sufficiently copious flow of blood has occurred. As you see, we are veering back to the old idea of the purifying effect of menstruation.

On the other hand, if ovarian secretion is produced in quantities insufficient to stimulate the uterus to menstruate, these symptoms seem to establish themselves more or less in perpetuity. Thus we find in many cases of oligomenorrhea and amenorrhea, and particularly in the climacterium, a syndrome in which frontal and temporal headaches and neuralgias of various cranial or peripheral nerves predominate. In some instances, astigmatism and other disturbances

of vision, carious teeth and the like may be the cause; but, as a rule, the regular occurrence of the symptoms mentioned points to the fact that chemically toxic substances are secreted by the ovary into the circulation, for which there is no outlet. These substances have a definite vasomotor action, as evidenced by the well known hot flushes in amenorrhoic and climacteric women. In the latter I have very frequently observed a phenomenon which I have never found mentioned in literature: a blinding headache which exerts an insufferable pressure just behind the eyes. If in such women, a tablespoonful or more of blood is taken from the uterus by scarification of the cervix, the headache disappears instantaneously, even before the patients rise from the examining table. Suggestion can altogether be ruled out, because the patients are not aware of the bloodletting. A venesection may, possibly, have the same effect; but of this I have no experience. These specific *climacteric* headaches occur in many patients quite regularly at intervals of from four to six weeks and always yield promptly to the same treatment, until the definite cessation of the ovarian secretion makes an end to this distressing symptom. *The great practical importance of this observation to the laryngologist and oculist needs no further comment.*

In the realm of obstetrics, headache is encountered as a conspicuous symptom in several conditions.

Many women present in the latter months of pregnancy marked changes of their facial appearance. The nose becomes broader, the lips thicker, the cheeks are more prominent and the ear lobes enlarged. Often, the fingers, too, seem to swell, so that rings have to be removed. With it, there is a steady and at times severe headache, and yet blood pressure and kidney function remain perfectly normal. We have to deal in such cases with a physiologic hypertrophy of the hypophysis due to pregnancy which, after childbirth, gradually resumes normal proportions, with concomitant disappearance of the symptoms mentioned. This *pituitary* headache, therefore, is not amenable to treatment.

Much more serious is another form of headache which occurs in pregnant women who also exhibit an increase in blood pressure, albuminuria with reduction of the urinary output, edemas, and disturbances of vision. Such women are in a pre-eclamptic state, which will terminate in true eclampsia with convulsions unless appropriate treatment is instituted without delay. This *pre-eclamptic* headache, which is one of the manifestations of a toxemia, requires no separate treatment; it disappears spontaneously if the suitable therapy has been successful in eliminating the poison.

Still another form of *toxic* headache is that found in cases of puerperal fever where bacterial toxins have entered the circulation. Its relief depends on the means employed to combat the existing sepsis.

Summary: As we now look back over the field just traversed, we realize that headache in women, though it is a very common complaint, has, in the great majority of instances, little if any relationship to the gynecological condition present. It appears in various types, which are the same in men and women, and may be produced by a multiplicity of causes.

Only in a comparatively small number of patients do we find headaches in which a direct casual nexus between them and the pelvic organs can be established. This list comprises in gynecology the headache of the menstruation, of amenorrhea, and of the climacterium; and in obstetrics the headache caused by hypertrophy of the hypophysis in pregnancy, by the pre-eclamptic state, and by puerperal sepsis.

As you see, the list is small but to us gynecologists it is very important, because in these few conditions the symptom of headache has pathognostic value. It may guide us towards the right diagnosis and thus to appropriate treatment.

For the very same reason this list is also important to the laryngologist, the oculist, in fact, to any specialist who may have under his care a woman complaining of headache, because the knowledge of the existence of specific gynecological or obstetrical headaches will prevent useless treatment or stay his hand from an unnecessary operation.

In exactly the same manner will the gynecologist be benefited by learning of specific headaches in other lines which this symposium has taught him. And thus, our conclusions must needs swing back to the thought expressed in my introduction, namely that the development of every medical specialty, after an extended period of separate growth, tends towards a readjustment, a reorganization with all the other specialties, and that the future of the art and science of healing lies in a Medical League of the Human Body.

Metropolitan Building.

HEADACHE AND NEURALGIAS OF NASAL ORIGIN.*

DR. ARTHUR M. ALDEN, St. Louis.

There are few diseases which may not at some time in their course have headache as a symptom. One sometimes suspects that most specialists, dealing as each does with a limited field, sincerely believes that most headaches are the result of disturbances within his own specialty. Even the laity speak with complete assurance of eye headache, sinus headache or headache due to constipation.

Of the patients who are referred to or who consult the rhinologist, the major part of them mention headache as one of their chief complaints. How many times have we all examined such a patient and having found some definite pathologic condition in his nose, advised an operation, with the complete assurance that when this was done he would at once and for all be relieved of his pain. The patient being convinced that we knew what we were talking about so confidently, the operation was done, with a beautiful postoperative result, except, that the headaches were just as frequent and just as severe as before. After several such experiences at the hands of men, each eminent in his own field and without any considerable relief of his pain, the patient often becomes disgusted and turns to Christian Science or the chiropractor for his result. It is in an effort to prevent just such failures that this symposium has been presented here tonight.

Inasmuch as headache is frequently a local manifestation of a remote systemic disturbance, a careful history may often be much more enlightening than an examination of some one particular region. That there exists a definite hereditary factor in susceptibility to headache is well established. The amount of disease that in one patient produces head pain to the point of nausea and complete prostration, may in another individual cause only slight discomfort. This apparent predisposition to headache on slight provocation is much more common in women than in men. On account of these factors and the many other variable considerations that have been so well elaborated for you in the preceding papers, it is perhaps unnecessary to stress the fact that before any serious surgical procedure is advised for the relief of headache, the patient should be carefully evaluated from every possible angle.

*Read before the St. Louis Ear, Nose and Throat Club, Dec. 21, 1927.
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Manifestly, such a thing as a pain in the head which is purely a clinical phenomenon cannot be subjected to an exact analysis, so that though volumes have been written on this subject, our information concerning the causative physiological mechanism and how it operates in the production of head pain is meager indeed.

Pain has been defined as the sensation produced by an injury to a nerve at its origin, in its continuity or terminal sense organ.

This injury may be the result of trauma, irritation or disease in the nerve elements themselves. Inasmuch as intrinsic disease of the nerves supplying the head is very rare, most head pains would therefore be due to the first two causes, trauma or irritation. The principal form of trauma to which the nerves supplying the nose may be subjected is pressure. So with this as a basis we may classify nasal headaches due to pressure into the following groups:

1. *Sinus Pressure Pain Due to: a.* Obliteration of the sinus lumen by mucosal swelling or closure of the normal ostium with retention of secretions and pressure upon nerve terminals in the submucosa of the sinus. *b.* Closure of the normal aperture of the sinus and absorption of the contained oxygen with intense suction upon and passive congestion of the venules in the sinus mucosa. This is the typical negative pressure sinus pain.

2. *Pressure Pain in the Nasal Cavity Proper, Due to: a.* Deformities of the middle turbinates or septum or both, leading to contact and pressure, which is greatly increased in the presence of congestion due to any cause. *b.* Pain caused by pressure from polypi or other new growths. *c.* Pressure upon nerves due to narrowing of the bony foraminae by hyperplastic processes.

Pain Due to Irritation: Under this heading belong the true nasal neuralgias, sphenopalatine ganglion and anterior ethmoidal. The pain in these is usually due to lowering of the threshold for irritation by familial predisposition or by disease of the nasal mucosa which affects the nerve terminals, the nerves themselves or the nasal ganglion.

In no other part of the body is mucous membrane subjected to the amount of insult, traumatic and irritative, that the nasal mucosa receives. The frequent coryzas, the structural deformities, the various infections tend to produce a region where the pathologic picture is always a complicated one. The above-mentioned pressure factors rarely act singly in the production of headache but more often the pain is due to several of them acting intermittently or at the same time. This explains in part the reason for the variations in the types of headache which may be the result of mechanical factors in the

nose. Even after careful study of the nose, the decision as to the exact importance of each of the factors which may be acting in any given case is usually difficult, and often impossible.

The nasal examination in such a case should be systematically carried out with the best possible means of illumination. After careful inspection, the effect of shrinkage, without cocain, should be noted. For this purpose I have found ephedrin hydrochlorid 1 per cent in watery solution most useful. Where cocain is used for this purpose, the effect of shrinkage upon the pain is masked by the anesthesia of the nerve endings. When the pain is completely relieved by shrinkage alone, we may conclude that the headache was the result of mechanical factors and it remains only to ascertain which of these are acting in our particular case. The fact that the pain was not relieved by shrinkage does not entirely rule out pressure, for occasionally the anatomical configuration of the lateral wall or the septum is such that it is impossible to put the shrinkage solution where it may be effective. Pain due to a closed empyema of a sinus, a closed sinus with negative pressure or a nerve pinched by a narrowed bony canal may not be relieved.

Cocain has two effects upon mucous membranes, a local anesthesia from paralysis of the sensory nerve endings or block to the nerve trunk or ganglion and an astringent action due to stimulation of the constrictor fibers in the muscle walls of the arterioles in the submucosa. When cocain is applied to the nose for diagnostic purposes, these two effects must be carefully differentiated, otherwise confusion as to its exact action and possibly diagnostic error will result. The effect of an application of cocain to the nasal ganglion or to the trunk of the anterior ethmoidal nerve has a very valuable significance. This procedure was first suggested by Sluder and has now become a standard diagnostic experiment.

The fact that a headache may be entirely relieved by an application of cocain to the region of the sphenopalatine foramen usually means that the pain is due to nerve injury in the ganglion itself or in the sensory fibers distal to it. Where pain is relieved by a cocain block applied to the trunk of the anterior ethmoidal nerve, Sluder believes that it is always diagnostic of anterior ethmoidal neuralgia. As the result of these procedures the diagnosis and the treatment of the nasal neuralgias have been placed upon a rational basis and the group of so-called idiopathic headaches have been further limited.

There are, however, several possible sources of error in this experiment. The position of the ganglion with regard to its proximity to the mucosa of the lateral wall of the nose is variable, due to the type of posterior ethmoidal pneumatization present. The location of

the sphenopalatine foramen is not constant with regard to the posterior end of the middle turbinate. The configuration of the septum or the structures on the lateral wall may be such that it is impossible to make an application directly over the foramen. I am sure that these factors are responsible for many of the failures to obtain definite results from these procedures. The fact that a headache may be relieved by an application of cocain to an appropriate part of the nasal mucosa does not always mean that the cause of the pain is nasal. For example, I have several patients in whom I can entirely relieve their premenstrual headache by a five-minute cocain block applied to both anterior ethmoidal nerve trunks.

The diagnosis of the site of the nasal lesion causing pain that is due to acute pathology is rarely difficult. The history, with the examination, all make a picture that is usually unmistakable. It is the chronic conditions where the location of the pain is inconstant and its periodicity less characteristic that sometimes tax our diagnostic acumen to the limit. We frequently see patients with advanced pathologic changes in the nose of both the suppurative and hyperplastic type who never complain of pain, while in others, a much less significant lesion produces intense suffering. Does the presence or absence of pressure alone account for the difference in these cases, or are these divergent reactions to the same thing dependent upon the fact that in certain persons the pain threshold is much lower than in others?

In the examination of such cases, we are apt to err in one of two opposite directions. The first is in the tendency to attach too much importance to some one local lesion and thus lead the patient into a serious surgical operation which may fail to relieve him of his symptoms. The second is the possibility of disregarding as unimportant a slight local change which may be the only visible sign of the disease condition that is really the cause of the pain. This last statement particularly applies to the sometimes slight changes in the posterior half of the nose, which are often the only observable signs of chronic hyperplastic ethmoiditis or sphenoiditis. I know of no rhinologic problem which requires greater nicety of judgment than the proper evaluation of these occasionally slight but most important signs.

The first requisite for the recognition of the visible signs of disease changes in the nose is an adequate means of illuminating the various recesses in the nasal cavity. For a proper examination of the vault of the pharynx where the light must be reflected from a mirror not more than 1 c.m. in diameter, some form of brilliant, white light

must be used. In visiting the offices of rhinologists in various parts of the country, I have seen the specialist squinting at a feebly illuminated posterior half of the nose and then heard him say that in his practice he rarely saw those interesting forms of sinus disease that have been so frequent among the patients of some of us in the Middle West. One does not need to seek far for the reason. Most of us in this part of the country use the arc lamp which has been popularized by Dr. Sluder. I know of no other form of light except direct sunlight that gives one such a brilliant illumination, not only of the configuration of the structures, but it makes visible even slight color changes in the mucous membrane. The nasopharyngoscope also makes certain regions, particularly the middle meatus, available for inspection.

Prior to the last two years, the X-ray has been valuable principally in the fact that it gave us a good view of the bony anatomy of our case rather than any adequate conception of the changes brought about by disease. With the advent, however, of lipiodol, the value of the X-ray in the diagnosis of sinus disease has been greatly enhanced. The introduction of lipiodol into a sinus, either by a direct injection or by the displacement method of Dr. Proetz and radiographs in various positions, now gives us information concerning intrasinus changes that hitherto had been obtainable only upon the operating table. It is now possible to say with all confidence before operation that a sphenoid ostium is tightly closed, or that the cell contains a large polyp or greatly thickened mucosa. Not that it is necessary or even advisable to apply all of these refinements in diagnosis to every case, but in the obscure cases they often afford us much additional information.

These patients with long-standing headache, accompanied occasionally by the profound mental depression that is only another symptom of the same disease that produces the pain, are certainly deserving of our best efforts. Their cases are always complex and a snap diagnosis with its operation too often results only in making the patient more difficult for the next specialist. No effort should be spared in making a careful, systematic, painstaking attempt to establish a definite diagnosis before any surgical procedure is suggested. We must learn to consider the patient as a whole rather than as just another headache case. This, and only this, will enable us to reduce the number of our surgical failures and keep more of these unfortunate people out of the hands of the cultists and the charletans.

HEADACHE.*

DR. DANA W. DRURY, Boston.

PART I.

Headache may be a comparatively independent affection, and remain so throughout the greater part of life. Although in such cases it may sensibly prejudice the enjoyment of existence and interfere in no small degree with the capacity for work, it is nevertheless in this form no dangerous evil, nor threatening to life. But here also, as we shall see, precise diagnosis and differentiation of individual varieties is by no means always easy, although these are essential preliminaries to the efficient course of treatment.

On the other hand, headache is one of the commonest symptoms of the most diverse complaints, acute and chronic, mild and severe. There is hardly an organ in the body damage to which is not accompanied by symptomatic headache. In such cases also it not infrequently dominates the scene, and often enough, if only temporarily, stands in the foreground of the whole clinical picture. At times it is one of the first signs of a severe disease, and on that account of no little prognostic significance. For all these reasons every patient presenting himself to the physician with the complaint of headache should be submitted to a minute and complete examination.

Not only the nervous system, but the organs of the thoracic and abdominal cavities, the pulse, blood, urine and much besides must be carefully investigated. It is not, however, on that account necessary to assume beforehand the existence of a serious complaint. It should always be borne in mind that so far as life is concerned the great majority of sufferers from headache may be given a good prognosis. In the present state of our knowledge any systematic classification of the different forms of headache offers considerable difficulties. Formerly a distinction was made according to the severity of the complaint; cephalgia, under which name were included the milder forms, and cephalea, which designated the more severe ones. Other distinctions were the accidental from the habitual, the transitory from the chronic, the periodical from the continual, the essential from the systematic.

An attempt was made to divide true headaches from allied affections, such as neuralgias, as well as from head pains in diseases of

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the throat and nose, and of the accessory sinuses, in dental caries and aural disease, from hysterical headache and the oppression of the heda of neurasthenics. True headaches were classified into those without obvious anatomical lesion and those caused by organic disease. Amongst the former were included: Migraine, the headaches of children, of adolescents, the headaches of anemia and vasoparalytic headaches. Amongst those caused by organic disease would include hyperesthesia of the hairy scalp, nodular headache, those due to disease of the bones of the skull or of the periosteum, also those caused by disease of the brain and its coverings.

It is possible that these attempts at classification are too one-sided, too superficial, possibly also partly too comprehensive, as were those of earlier times; thus the classification mentioned above, however desirable, goes ahead of the facts at our disposal, takes insufficient account of our practical requirements, and fails to attain its aim. It is by no means decided whether there may not be anatomical changes underlying migraine. The headaches caused by dental caries, diseases of the nose and throat and ears have also underlying organic changes, which lead to irritation of the dural nerves. Hyperesthesia of the scalp occurs in all sorts of functional and organic headaches. There are also good grounds for doubting whether it is feasible to distinguish the headache of children and adolescents as a special subdivision of the group characterized by absence of an anatomical lesion. It is obvious that, in the present state of our knowledge upon this subject, it is not even remotely possible to undertake a classification upon an etiological or anatomical basis. That this must be the goal of our endeavor is beyond dispute.

Even although the fact must be emphasized that headache, pathologically speaking, has no individual entity as a disease, it would be contrary to facts to deny that this symptom very frequently persists a long time as the sole or most prominent one, and must be regarded as an entity from the point of view of differential diagnosis. It is not long since every headache was considered to be of nasal origin and was treated as such. We have learned with time to free ourselves of such exaggerations and to place differential diagnosis upon a more precise basis. It stands today established that headache in nasal disease of every sort is an extremely distressing symptom and may, on account of its severity and obstinacy, mask all others.

That diseases of the nose are capable of producing headache should cause no surprise, since the sensory supply to the region in question is entirely from the first and second branches of the fifth nerve. The anterior and posterior ethmoidal nerves springing from the

frontal branch of the first division (ophthalmic) innervate the mucous membrane of the anterior part of the nasal cavity, the septum, the anterior ethmoidal cells and the frontal sinus, as well as the skin of the nose as far as the tip, and that of the forehead from the middle-line as far as the center of the orbital margin. The second division (maxillary) gives off the superior dental nerves supplying the floor of the nose and the maxillary antrum; in addition sensory twigs go from the sphenopalatine ganglion to the mucous membrane of the posterior portions of the nasal cavity and septum, as well as to that of the posterior ethmoidal cells and sphenoidal sinus. These three sensory zones are therefore of importance because a consideration of them affords a clue to the location of nasal diseases.

Speaking generally, localized pain at the upper inner corner of the orbit or over the eyebrow denotes disease of the anterior part of the nasal cavity, of the frontal sinus, or of the anterior ethmoidal cells; pain situated deeply in the head, to disease of the posterior part of the nasal cavity or of the sphenoidal sinus, and pain in the cheek or in the teeth (dental trouble being excluded), extending to the nose or cheek, points to disease of the antrum or floor of the nose.

Although these local and neuralgic pains of nasal origin are, strictly speaking, not to be reckoned as actual headaches, they must nevertheless be discussed for diagnostic reasons, quite apart from the fact that in individual cases pains in the interior of the skull occur as well, as for instance, in empyema of the frontal sinus. Localized pains occur almost exclusively in acute inflammations of the accessory sinuses. In acute empyema of the frontal sinus and maxillary antrum there is often in addition a localized tenderness to pressure of so acute a nature and anatomically so exactly corresponding to the position of these cavities that without more detailed examination (transillumination, etc.), the diagnosis can easily be made.

In the acute inflammations of the ethmoidal cells and sphenoidal sinus a careful examination is naturally essential. The spontaneous pain may also radiate into surrounding parts, as from the antrum to the teeth and temporal region, from the frontal sinus to the eye and forehead as far as the margin of the hair; it must also be borne in mind that sphenoidal sinus pain is often referred forwards to the forehead. The extraordinary acuteness of the pain characteristic of these cases is, in addition to the pressure upon the trigeminal nerve-endings through inflammatory swellinf of the mucous membrane, to be explained by the simultaneous pressure effect due to retention of exudate in the cavities owing to closure of their orifices.

Neuralgiform pains in the course of the supra- and infraorbital nerves and of the superior dental nerves occur with the acute and

chronic inflammations of the accessory cavities, partly owing to direct extension of the inflammation (perineuritis) to the nerves in relation with the particular bony walls (supraorbital with that of the frontal sinus, infraorbital and superior dental branches with that of the antrum), partly through irradiation to another branch. This latter mode undoubtedly accounts for the neuralgia in the region of the supraorbital nerve which sometimes accompanies inflammation in the antrum, since it is well established that even intermittent frontal headache may be present with disease of this cavity without any involvement of other accessory cavities (frontal or sphenoidal).

Actual headache referred to the interior of the skull is more or less widely diffused over the whole head. Initially and in the milder cases it is a pressure, resembling the neurastehnic, which may increase after a time and become a dull pain, primarily in the forehead, but also affecting the vertex and occiput. A universally familiar example of this is to be found in the mental obfuscation of an acute cold with the associated characteristic aprosexia of the nose (incapacity to fix the thoughts or concentrate the attention), which may serve as a prototype of all those headaches which arise as a result of inflammatory swelling of the mucous membrane and nasal obstruction. Their pathogenesis is to be sought in the passive hyperemia accompanying these nasal affections, which on anatomical grounds spreads to the interior of the cranium.

The veins of the nasal cavity communicate extensively with those of the brain, dura and longitudinal sinus and the nasal lymphatics by means of the lamina cribosa with the great subarachnoid and subdural lymphatic space. Thus a stasis both of blood and lymph circulation may ensue. This congestion is further increased through the fact that at each breath, as the result of a rarefaction of air on the obstructed side, fluid is aspirated into the upper air passages. The diminised or suspended nasal respiration has a further prejudicial effect, in that the absorption of oxygen in the circulation is lessened as well as the elimination of carbon dioxid and other toxic products of combustion. Thus a diminution of hemoglobin (anemia or, more correctly, chlorosis) is brought about in the brain and its coverings as well as in the rest of the organism. That these sequelae of nasal obstruction cannot be got rid of by means of drugs is obvious. They occur through relaxation of the alae nasi, particularly seen in old age, in chronic rhinitis, often leading to hypertrophy of the posterior ends of the inferior turbinates, in chronic inflammation of the accessory sinuses, in hyperplasia of tonsils, adenoids and ozena, also with polypi and all forms of tumors of the nose.

According to my observations there is in the last class of cases early hyperesthesia of the cheeks and the skin of the nose, particularly when the tumors originate in the sphenopalatine fossa. There can, too, no longer be any doubt that deviations and thickenings of the septum, often of traumatic origin, bony ridges and spurs of the same, either alone or in association with the above mentioned affections, if they lead to narrowing of the nasal passages or contact between the septum and the turbinates, may give rise to such forms of headache. These headaches, however, are not persistent, lasting only for a short period and while the pressure remains.

In rarer cases the latter may arise not only by means of the above mentioned circulatory disturbances, but also reflexly, for the most part only in association with general hyperesthesia, as is characteristically seen in the clinical picture of neuresthenia. The stimulus in such cases may come from any part of the nose; there is then usually considerable hyperesthesia on examination with a probe, the richly innervated tuberculum septi, from which, as is well known, asthma attacks may be precipitated, being especially susceptible.

Since there is an intimate relationship between the nasal mucous membrane and the genital organs, and a reflex nervous hyperemic swelling of the mucous membrane may often be observed during menstruation (or with strong sexual excitement), the assumption is not untenable that the exacerbation of headaches of variable origin so frequently occurring at the menstrual epoch and the periodic reccurrence of migraine attacks precedent to or coincident with it may in part at least be the result of this nasal obstruction. It must also be admitted that the migraine tendency—quite apart from any connection with the sexual organs—may be brought to a head by any pathological process leading to nasal obstruction.

In the diagnosis of nasal headache, besides the above points the following must be taken into consideration: Its recognition is most easily affected through the characteristic localized pain, associated with clearly defined tenderness on pressure. The neuralgic pain offers greater difficulties. The following are the commonest causes of typical tic: malaria, rheumatism (arthritis), ocular troubles, pulp stones, influenza and intoxications. Influenza is of particular importance since both inflammations of the accessory sinuses and genuine neuritic neuralgias are common events, so that only detailed examination can decide between them. The statement made from the rhinological side that every neuralgia in the neighborhood of the fifth nerve occurring after influenza is the result of an acute sinus is open

to question, as is evident from the frequent cures of these neuralgias by means of simple antineuralgic treatment, leaving the nose entirely out of consideration.

The distinction from migraine is always easy if the hereditary nature of the latter and its commencement in early life be kept in mind, together with the realization of the fact that vomiting and similar phenomena representing aurae are not accompaniments of nasal headaches. Since, as we have already seen, inflammatory ocular affections may be associated with headache, and, moreover, as we now know, diseases of the nose and accessory sinuses may to a considerable extent implicate the eye (retrobulbar neuritis), so much so that patients may first consult an oculist, great care is essential lest the mistake be made of treating the secondary effect rather than the cause. Confusion with rheumatic headache is most readily avoided by carefully palpating the muscles of the scalp and back of the neck. The commencement of nasal headache in the occipital region, as is the rule in nodular and induration headache, is extremely common.

The greatest diagnostic problems are likely to arise on account of the resemblance between neurasthenia and nasal headache. In both there is mostly a sense of pressure. Only in a careful history, together with the findings within the nasal chambers, especially the frontal sinus region, settles the question. Inquiry concerning discharge from the nose, obstruction of one or both nostrils, and disturbance of the sense of smell must not be omitted. It is also imperative that the possibility of a combination of nasal headache with any other form be borne in mind. In such a case the cure of the nasal condition will be followed by persistence or speedy return of the headache. The fact must in addition be emphasized that headache may be entirely lacking in the clinical picture of nasal disease, even the most severe, and that long-standing inflammations and tumors may run an entirely painless course. Therefore, one should guard against assuming every positive discovery in the nose to be the cause of a co-existent headache. Again, hysterical headache, much more frequently than neurasthenic, assumes a neuralgiform character, mostly in the guise of neuralgia of the supraorbital and occipital nerves, which are at times extremely difficult to distinguish from real neuralgias, since the typical tender points are also present. In keeping with its pathogenesis, however, and often in the highest degree, it is responsive to suggestive treatment, whether hypnotic or waking.

In diseases of the teeth neuralgias in the course of the maxillary and mandibular nerves may occur in addition to local pain. If the

teeth of the upper jaw, particularly the molars, are affected, there is often a severe, dull and boring pain in the corresponding temple, which may often be confused with other forms of headache. Therefore, one must bear in mind that not only dental caries, which is at once recognizable, but also diseases of the pulp, with an intact surface, may give rise to neuralgia and temporal headache, as may also in unerupted third molar. Why in the one case only a localized pain, restricted to the diseased spot, should be felt, and in the other a widespread neuralgia or an obstinate pain in the temporal region, without the nature and extent of the dental condition affording any adequate explanation, cannot be said with certainty. Apart from individual hypersensitiveness, which cannot always be substantiated, there must probably be variations in the anatomical course of the anastomoses which one feels tempted to advance as explanatory of this variable behavior. On the other hand, we may assume definite treatment, which is enuncleation of the tonsils, in a type of protracted headache, seen in chronic follicular sore throat associated with neurasthenic manifestations (readily produced fatigue, disturbance of sleep), and which may culminate in nocturnal sweats.

As to headaches in diseases of the ear, it is mainly in acute otitis media, both catarrhal and suppurative, that headaches, generally of great intensity, occur; these are at first confined to the corresponding side and are aggravated by the movement of the lower jaw in mastication. If in the suppurative middle ear disease perforation of the drum does not soon occur with evacuation of the fluid, the pains radiate all over the head, and are then of a throbbing and pulsating character. In these affections there is always a considerable tenderness on pressure in the region in front of the tragus and on the mastoid process; the otitic headache can usually be recognized by the above peculiarities, and also by the fact that fever is generally present, at least until the drum membrane ruptures or is incised. If the suppuration involves the mastoid process this also becomes the seat of spontaneous pain. The intracranial complications dependent upon acute and chronic disease of the tympanic cavity, to the latter of which belongs cholesteatoma, are frequently ushered in by severe headaches, in that case usually accompanied by tenderness on pressure and percussion, and are soon followed by other characteristic symptoms. Every protracted or severe headache should induce one to examine the ears and to inquire concerning former ear disease. The syndrome-complex of Menieré usually associated with disease of the internal ear (vertigo, tinnitus, vomiting), is generally accompanied by diffuse headache.

PART II.

Many times we find that a patient does not do as well as we expect; on further study, we find what we thought insignificant symptoms at first taking their position at the front. As it is generally accepted that the internal secretions of the ductless glands are in some obscure way correlated with the general condition of the organism, then for present purposes it may be sufficient to postulate that in certain measure the general metabolism of the organism is regulated by these secretions.

For illustrative cases bearing on these points I desire to mention briefly the following case histories. There are many facts, established and proven by experiment, which have formed the basis of certain objective methods for the determination of the functional capacities of the several glands composing the endocrine group (Rowe). Were it not for the fact that the performance of the necessary tests is so time-consuming, complex in detail and requires so extensive an instrumental equipment, the examination of these processes as aids to the diagnosis of the endocrinopathies would have a much wider application.

Pituitary Disease. Case 1. The patient was a poorly nourished man, age 44 years, presenting as his chief complaint a paroxysmal headache, left frontal, temporal and occipital. The condition started several months ago, with a sudden onset of severe pain in the left shoulder, leg and left hand. Arm and leg somewhat contracted and the vision blurred. In two days recovery with recurrence in seven weeks without paralysis. There had been some headache for over a year past. The patient is intermittently irrational and during attacks is in great fear of death. The memory has also become impaired. Has had occasional vertigo and an indefinite tinnitus. The headache is always frontal, temporal and occipital and of a boring nature. The physical examination shows poor teeth, sluggish knee jerks, left Babinski and Oppenheim, plantar reflex on left, ankle clonus present, abdominal reflexes sluggish on left. Marked pilosity is recorded, but as patient is an Italian this is robbed of much of its significance. The urine shows a somewhat scanty elimination, but this undoubtedly is referable to the patient's low food intake, a trace of acetone in one specimen confirms this idea, one specimen shows an s.p.t. of albumin and one "urobilinogen". Beyond a rare hyaline cast the sediments are negative. The nitrogen partition confirms the inference regarding the food intake and shows a normal formula. The phthalein test is normal. Sugar tolerance is also normal. The blood morphology shows a leucocytosis with a preponderance of the neu-

trophilic elements, and there is a 4 per cent eosinophilia. The serologicals are negative. As the patient was bedridden his weight was calculated and he is somewhat below the weight predicted from his sitting height. His lung capacity is about half the calculated amount. The basal metabolism shows a practically normal rate with slow pulse and low blood pressure, Alveolar CO_2 was normal as was the salol test. The X-ray showed left frontal and antrum slightly obscured, large diploic veins and erosion of the posterior walls of the sella. The eyes show papilledema of both nerve heads, signifying intercranial lesion.

Diagnosis: The patient presents a pituitary tumor with resultant intercranial pressure. While certain of the characteristic findings of pituitary disease are recorded, the functional level of both lobes at the time of observation is substantially normal. The gland presents a marked hyperplasia without change in function.

Case 2: The patient was a tall, slim woman, age 44 years, with a somewhat protruding and noticeably square lower jaw. Her presenting complaint is of headache, from which she has suffered for 20 years, with an increasing severity up to eight years ago, since when they have abated in intensity but have increased in frequency. At onset, the attacks appeared at intervals of six weeks or more and lasted from one to several days. More recently the interval has shortened to one or two a week. Eight years ago at the period of greatest severity, she developed sinus trouble, requiring operation. X-rays taken at this time disclosed a pituitary tumor.

The headaches begin over one eye and gradually extend over the whole upper half of the skull. Heat or cold may bring some relief. The family has shown much cardiorenal disease and there are cases of tuberculosis and cancer in near relatives. During the last few years, the patient has been under a severe nervous strain nursing her invalid mother and sister, both of whom died recently. Her own history records the minor ailments of childhood, scarlet and rheumatic fevers and several attacks of influenza (one with pleurisy) in later years. She was undersized until the age of 16, at which time she established the catamenia and grew very rapidly. The menstrual history is substantially normal. Always has headache at end of period. Other pertinent points in the history are photophobia, some vertigo and recently some degree of nervous instability.

The physical examination shows a protruding and noticeably square lower jaw, sallow complexion, infected, ragged tonsils, a friction rub over upper half of left lung, possibly palpable left kidney, slight pilosity of legs and very active knee jerks. The hands and

feet are not enlarged. The urine shows a normal volume, fair elimination, and contains albumin and occasional hyaline cast, with renal tubule and bladder cells presenting evidence of renal irritation. The nitrogen elimination shows a protein intake below a maintenance level. The residual nitrogen fraction shows a borderline high value. The phthalein test is below the normal, the galactose tolerance somewhat low; the urea curve is of the flat type with poor elimination. The blood morphology shows a fairly normal blood with slight lymphocytosis. The blood chemistry shows low values for all nitrogenous constituents, but the blood uric acid is relatively high. The serologicals are negative. The patient is 20 per cent under weight, with normal lung capacity. The basal rate is recorded as —4 per cent,* with slightly slow pulse and a definite low grade hypertension most marked in the diastolic pressure. A nose and throat examination confirms the tonsil condition. An X-ray shows some thickening of the membrane in the antra and very definite evidence of a pituitary tumor of a slow growing type. The eye examination shows yellow discs.

Diagnosis: The picture here presented is one of a mild degree of pituitary dysfunction with the anterior lobe under- (lowered basal rate) and the posterior over-active (lowered galactose tolerance), associated with hyperplasia of the gland. The present status in conjunction with the history implies a probable earlier hyperactivity, which is now undergoing functional involution, in which latter the anterior lobe has already reached a hypofunctional state. This determines a fairly common formula of pituitary disease. A point of interest lies in the fact that the patient's eye tests are not comparable with the severity of the presenting symptom if intercranial pressure were the sole etiological factor.

Case 3: The patient is an obese elderly woman, age 71 years, with marked loss of hearing but otherwise outwardly well preserved. Her chief complaint aside from the deafness is of severe, dull, boring headache which first began five years ago. She relates that on stooping over something seemed to snap in the head. This was followed by a severe headache lasting three days. There was no recurrence for over a year and then the headaches recurred at first at intervals several months apart but with progressive ferquency so that during the last year they have come on an average at three-week intervals. A typical attack is described as follows:

"The pain started in the night without warning and lasted about 12 hours. It was a severe, deep, boring ache. She at once took pow-

*This value is certainly well above the truth, as the patient was nervous and somewhat restless.

ders recommended by her local physician (some form of narcotic drug, the nature of which could not be ascertained) and ultimately this medication helped the condition."

The sensation is always deep in the head, begins over the frontal area, extends to the occiput and is a boring, violent, thumping sensation. Following the attacks she feels exhausted and sleeps many hours if undisturbed. The attacks are never accompanied by nausea or vomiting and she is never dizzy. Polyuria always accompanied the headaches. The family history shows several cases of cardiorenal disease. The patient's own history records a nervous breakdown in 1895, necessitating a residence of seven months in a sanitorium. The right breast was removed 20 years ago because of a benign tumor and she had a pelvic operation, probably a ventral suspension, somewhat earlier. Has only lost six teeth. Her menstrual history is essentially uneventful. Physical examination shows marked senile skin changes, very marked deafness in both ears, the presence of much dental work, an apparently normal heart, negative lungs, Heberden's nodes prominent in fingers and knee jerks hard to elicit. The urine shows a normal volume and an elimination which is fair, considering the patient's age and dietary restriction. Her protein intake is at a somewhat low level. The galactose tolerance is well above the normal, a positive response being elicited first with 60 gms. As the postmenopausal level lies in the neighborhood of 30 gms., this is an increase of nearly 100 per cent. The urea curve is normal, although the elimination is only fair. The blood morphology shows a slight secondary anemia and there is a definite lymphocytosis. The blood chemistry shows a high uric acid with the other nitrogenous constituents at a normal level. She is 14 per cent overweight and shows a significant reduction from her predicted lung capacity. The basal rate is recorded as —11 per cent, with a second observation at —15 per cent. The pulse and blood pressure are both normal for one of the patient's age. The orthopedic examination shows the postural curve of old age with a compensation lordosis; the joints show characteristic hypertrophic spurring, but are otherwise negative. X-ray plates confirm these latter findings, show the right mastoid cells to be obscured and demonstrate an erosion of the posterior wall of the sella. The eyes show yellow discs, slightly enlarged blind spots, and some cutting of the form fields most marked on the right side.

Diagnosis: This case is one of bilobar pituitary failure with hyperplasia of the gland.

Comment: A characteristic of these headaches is that they usually stop at once on lying down, to begin again on the upright position.

Generally they are very disabling and persistent, but they are also paroxysmal. They are of a boring type, usually bitemporal, and very intense. They are usually sensed as confined within the head and tend to cause the patient a good deal of drowsiness. Every pituitary case, however, does not have headache and occasionally as a symptom it is never mentioned.

Thyroid Disease. Case 4:. This patient was a young woman, age 30 years, presenting the characteristic picture of well marked exophthalmic goiter. While this was naturally her chief complaint, she emphasized headache as one of her most distressing symptoms. These headaches began in early childhood; they usually start in the frontal region, left or right, and extend throughout the entire head; stooping or bending increases the severity. The attacks are persistent, cause a confused feeling and oblige her to lie down, preferably in the dark. With an attack vertigo is usually present, but this varies much in severity. Commonly, the room seems to oscillate in all directions; there is marked tinnitus and a lessened acuity of hearing. The family history shows malignant neoplasm in the mother and one brother. The patient's own history is not remarkable beyond the account of the progress of her thyroid disease, which was first noted nine years ago and has since been steadily progressive. Loss of weight, dyspnea, tachycardia, tremor, nervous instability, menstrual irregularity and gastrointestinal disturbances are all referred to the thyroid disease. Physical examination shows good development with some emaciation; the patient is extremely nervous, hair on the vertex is dry and thick, there is very marked exophthalmos, the tongue shows tremor on protrusion, many teeth are missing, tonsils are enlarged and cryptic, and a bilateral enlargement of the thyroid gland, the heart shows several abnormalities, including a tachycardia which reached 186 beats per minute, her blood pressure at the time of the examination was recorded as 190/80, which was certainly a reflex of her profound nervousness; (her systolic level at the time of her basal test was 135:) a slight enlargement of the liver was recorded, the tendon reflexes were exaggerated and there was marked tremor of the hands, with an occasional coarse tremor of the head. The urine volume is scanty, elimination inadequate. A slight trace of albumin and increased indican are reported, while the sediment is negative. The protein intake is definitely below maintenance, and in view of the patient's hyperthyroidism is entirely inadequate. The formula shows a slightly high residual nitrogen. The blood shows a slight secondary anemia, with normal leucocyte formula. A high borderline uric acid is the only abnormal feature of the blood chemistry.

The serologicals are negative. The phthalein test is a low borderline value. The patient's sugar tolerance is much below the normal amount. She is 14 per cent underweight and has lost nearly two-thirds of her predicted lung value. Alveolar CO_2 is normal. The basal rate is recorded as +47 per cent, a fair average value in comparison with other records. The blood pressure is normal; the pulse varies from 84 to 132. Cardiogram shows auricular fibrillation. The patient shows yellowish discs, the fields ars markedly and symmetrically contracted, and the blind spots are enlarged.

Diagnosis: The primary diagnosis here is exophthalmic goiter with hyperactivity of the gland and a related cardiac condition. An interesting feature is found in the two anomalous observations of the markedly low sugar tolerance and greatly contracted form and color fields. Hyperthyroidism usually produces only a very moderate drop in the galactose tolerance. In the present case the two anomalies are referable to a concomitant liver disorder. As is well known, this produces a marked lowering of sugar tolerance; marked symmetrical contraction of form and color fields has been observed in an extended series of liver and gall bladder cases.*

Case 5: The patient was a man age 47 years, complaining of frequent attacks of light-headedness similar to vertigo but differentiated, in that he does not deviate from a straight line in walking and closing the eyes does not lessen the sensation. The attacks have been progressive in frequency, they are usually ushered in by an increase of a whirling sound in on or both ears and may appear at any time. The mother died of cancer. The patient presents a long history of gastro-intestinal disorder with an appendectomy three years ago, and the identification of a duodenal ulcer which has apparently been corrected by dietary regulation within the last year has had much trouble with nasal obstruction with operative intervention which has been only partially successful. There is also an ill-defined asthmatic tendency. Physical examination was entirely negative. The nitrogen partition shows a normal protein intake, the residual nitrogen fraction is above the normal. The blood morphology is not remarkable except for a slight lymphocytosis which equates with the high residual nitrogen. The blood chemistry is normal and the serologicals are negative. The phthalein test is distinctly below normal although the curve of elimination conforms to the convention. The patient's galactose tolerance is definitely lowered he showing a trace of sugar with a dosage of but 10 gms. He is 2 per cent underweight by the Dreyer standard, but his sitting height index is below normal. This would imply that the underweight is actually in excess of recorded figures. Urobilino-

*Unpublished data. Personal communication from Dr. A. W. Rowe.

gen is negative. The basal rate at the time of the examination is —2 per cent, but earlier records show a variation above +20 per cent. X-ray plates of various parts of the body, including a gastro-intestinal series, were negative, as was a neurological examination and a series of endermal tests. The eye examination showed yellow discs; a nose and throat examination demonstrated infected tonsils. A duodenal function test showed some biliary disturbance, while the Graham test by the intravenous route was negative.

Diagnosis: The patient's past history and current findings define a mild dysfunctional state of the thyroid following an earlier hyperactive condition. This condition is frequently associated with focal infective processes, and in the present instance the tonsils were seriously infected. In addition, a dysfunctional state of the liver was demonstrated, to which may be referred the abnormal sugar tolerance. At the time of the first observation the thyroid was in a transitional state of functional involution. Later measurements demonstrated a clear-cut progress toward terminal hypofunctional condition.

Case 6: This patient was a youth age 16 years, tall, well nourished. He has had trouble with both ears, lasting from two to seven days, every four or five weeks since childhood. It followed an early attack of pneumonia in infancy. During the past four or five years the attacks have been accompanied by pain and tenderness. As the attack terminated the exudate from the canals increased in amount. Intermittently through the years he has had a sense of pressure, at times giddiness and tinnitus. However, the tinnitus was not associated with the activity of his ear canals. The headache was not localized in any part of the head; it often gave him an expression of lassitude and depression. He expressed the sensation aptly as a general heaviness throughout the head, wishing that he might only rest his head on something soft. The lassitude and depression have been greatly helped by several tonsillectomies. He had the usual children's diseases, four attacks of pneumonia, and is very susceptible to colds. While the boy is entering college this year it has been very difficult for him to keep appointments on time and to acquire the study habit. The physical examination shows a dry skin, some dandruff, enlarged turbinates and thickened, red lateral walls of the pharynx. Both ears are occluded with exudate and skin, the underlying parts red and tender and bathed with moisture. Both drums negative, as are the mastoids and middle ears. He shows a normal audiogram for both ears. Many comedones in both conchas and about the auricles. The urine volume is normal, elimination good, balance fair. Protein intake is adequate, nitrogen partition normal, one specimen shows albumin,

the sediment negative. Blood morphology shows a definite lymphocytosis. Blood chemistry is normal. Serologicals are negative. The phthalein test is slightly below normal. The galactose tolerance is definitely depressed. The patient is 10 per cent underweight and with a normal lung volume. Two confirmatory basal tests show an average rate of —18 per cent and a slightly slow pulse. X-ray of the sinuses and ears are negative. An eye examination is normal except for marked cutting of the upper fields due to lid drop. A cardiogram shows a normal heart, while an abdominal examination shows nothing of note. A duodenal function test showed some biliary disturbance.

Diagnosis: The pathology in this case is certainly not clearly devined. The low basal rate, a low sugar tolerance and a lymphoid blood are the principal departures from normal. These would be consistent with a pituitary dysfunction, but none of the other findings are confirmatory. The cutting of the upper fields, together with a positive liver dysfunction, resolves the case to a thyroid failure, coupled with disturbance of the biliary system. This combination of a liver dysfunction, coupled with hypothyroidism, is being frequently remarked in this clinic. The relative causality is under investigation.

Comment: In Case 4 there is an exophthalmic goiter with hyperthyroidism, while Case 5 is a thyroid dysfunction in a transition state, and Case 6 one of hypothyroidism. Each of these cases shows active liver dysfunction as well. The headache is usually an earlier symptom in hyperthyroidism. In general it is migrainous in character, usually accompanied by giddiness and tinnitus. It is also very often accompanied by attacks of depression, as well as by attacks of excitation.

Gonad Disease. Case 7: The patient was an obese young girl, age 19 years, an orphan, presenting behavior problems involving dishonesty and perverse sexuality. Had been earlier diagnosed as a case of constitutional psychopathic inferiority, had been committed, and at the time of the examination was on parole. She complained of a constant dull sensation throughout her head, but generally it seemed to concentrate at the nape of the neck. With the dull, heavy feeling constantly present there are times when she has neuralgic flashings that include the entire head, and then nausea and vertiginal feelings follow. The family history is negative. The patient's own story records only minor ailments, a tonsillectomy; the remaining history, including that of the catamenia, is not significant. The physical examination shows a rather gross general development and facial expression, an obesity more marked in the body than the extremities,

teeth are moderately carious, a chest which shows some flaring at the costal margins with flattening of the normal thoracic curve, marked septal deviation, a slight inguinal adenopathy, and acniform eruption on face and chest. The patient is somewhat slow mentally, but not markedly unintelligent. The urine gives a generally normal picture, the elimination and balance being good. Nitrogen partition is normal. Phthalein test is slightly below normal. The patient is positive with 20 gms. of galactose, a definitely decreased tolerance. Urea curve is normal in outline but elimination is poor. Urobilinogen is negative. The blood morphology shows a slight relative lymphocytosis. Blood chemistry is normal, the blood sugar being a low normal. Serologicals are negaive. The patient is 23 per cent overweight. CO_2 is normal. The basal rate is reported as +5 per cent, with a silghtly febrile temperature and patient slightly restless. The blood pressure at time of this test is somewhat higher than in the other independent measurements and this and the febrile temperature indicate that the observed basal rate is certainly above the truth. The pelvic examination shows sparse pubic hair and is otherwise negative. The Barany test shows an almost total lack of response to stimulation, which is probably of congenital origin. A neurological examiation confirms the earlier diagnosis on constitutional psychopathic inferiority. X-ray shows a deep sella which is not significant. Eye examination shows only yellow discs, otherwise negative.

Diagnosis: The general picture here is consistent with a primary functional ovarian failure. Neither the normal menstrual history nor the patient's sex delinquencies are incompatible with this condition*

Case 8: This patient was a woman, age 51 years, both of whose ovaries had been removed 16 years before. She had suffered from headaches more or less constantly for over 20 years. They are generalized throughout her head, usually being more intense during the morning hours. During the beginning of a seizure she is troubled with attacks of weakness more or less throughout her body. At other times the attacks seem to start in either the left or right ear, with an intense surging, whining sound, together with slight numbness in her legs; she feels giddy and has the sensation of floating into space. Her sleeping, which is usually very good, is always disturbed during one of these attacks. Prior to her bilateral oophorectomy she formerly noted that the attack was accentuated at the menstrual period. The family history shows nothing but a rather frequent incidence of asthma, from which the patient also suffers. In addition to the operation noted above she records only minor ailments. Physical exam-

*The patient has shown marked clinical improvement and a normalizing of the laboratory picture under ovarian medication.

ination shows an emaciated and poorly developed woman, slight flushing over the malar bones, eyelid tremor when closed, teeth in good condition but show much dentistry, a long narrow chest with small breasts, hyper-resonance over most of the thorax, slight lumbar scoliosis. The urine is scanty in volume, the elimination is poor; small amounts of albumin and increased indican are recorded. Both sediments show many leucocytes, and in one fatty casts are observed. The nitrogen partition is normal, considering the low level of nitrogen elimination. The phthalein test is definitely below the normal level. The patient's galactose tolerance is half that of the normal, which would accord perfectly with her history of ovarian operation. The urea curve is of the progressive type, which speaks more for a pyelitis than for a nephritis. Urobilinogen is negative. The blood morphology is substantially normal in respect to erythrocyte count but shows a lymphoid type with a 7 per cent eosinophilia. This latter again is unquestionably referable to the patient's asthma and without any endocrine significance. The blood sugar is a low normal level. This is interesting in the light of the patient's extensive adrenalin medication. The Wassermann is negative. She is 33 per cent underweight and her vital capacity is recorded as 63 per cent below prediction. Two basal determinations show a consistent rate of —16 per cent, a low blood pressure, practically normal pulse and temperature. Endermal tests show the patient negative to a number of substances of common contact. X-ray examination shows a long, narrow thorax, increased desnity and mottling of both lungs, a right lumbar scoliosis, dislocation of the left hip, obliteration of the right sacroiliac, and several retained roots. The eye examination is substantially negative; there is a very slight contraction of the fields, which is probably not significant.

Diagnosis: The present condition is a clear-cut hypogonadism, initially functional in character, but for the last 16 years surgical in origin.

Comment: Both of these cases are ovarian failures. The first, purely functional in character, while the second derives from castration. The possibility of a causal nonendocrine condition in the second case makes it a somewhat less satisfactory illustration than the other cases here reported.

Migraine of Nonendocrine Etiology. Case 9: The patient is a fairly well developed man, age 48 years, who from early childhood has had severe left unilateral headache, starting in left supraorbital region and radiating along whole left hemicrania, followed by nausea and vomiting, the attack lasting up to 36 hours. Attacks are typical of

migraine, and eight other members of the family have it, only one child having escaped. Attacks are more frequent and more severe than formerly, occurring about every two weeks. During the attack there are throbbing pains in the head synchronous with the heart beat. Head feels heavy and dizzy about two days before the attack. Nothing in particular relieves, and there are no eye or ear symptoms. The vomitus is very sour and consists of food and bile. For the past ten years has had also attacks of epigrastric pain with marked constipation, and these attacks were so bad that eight years ago he had an exploratory lapratomy with negative findings, except that the appendix was removed. The physical examination shows a moderate degree of baldness and rather abundant body pilosity, patient is jaundiced, eyes are rather prominent with a slightly yellowish tint to the conjunctivae, tonsils irregular and cryptic. The family history shows several cases of cardiorenal disease and one of cancer. The patient's own history is devoid of significant facts, other than those given above. Urine shows normal volume, low elimination, and is otherwise negative. Nitrogen partition normal, with perhaps a slight upward tendency of the residual nitrogen. Blood morphology shows lymphoid blood with a 4 per cent eosinophilia and slight basophilia. The blood chemistry is normal, as are the serologicals. The phthalein, CO_2, and vital capacity are all normal. The basal rate is —16 per cent, with lowered blood pressure. X-ray of the skull was negative except for undeevloped frontal sinuses. The neurological examination was also negative. The sugar tolerance shows a marked depression, giving a positive response with 10 gms. The eyes show a slight enlargement of the blind spots . An intravenous Graham test was negative. A complete duodenal test (McClure) gave marked evidence of disturbed liver function.

Diagnosis: This case presents no evidence of endocrine disease, the lowered sugar tolerance being referable to the liver condition. The diagnosis is a liver dysfunction.

Case 10: The patient was a well developed, obese woman, age 47 years. For 30 of her 47 years she has had intense, disabling headaches, first beginning in the occipital region, but during the last five years involving the entire head, although most severe in the frontal and parietal regions of the right side. They seem to be especially severe at the menstrual time and of greater frequency. There seems to be a constant sensation of swelling and pressure inside of her head. They frequently awake her in the morning and many days last throughout her waking period. There are times that there is a good deal of fatigue with the headaches. The family and marital

history are not significant. The patient records minor ailments, recurrent tonsillitis finally corrected by operation, inflammatory rheumatism seven years ago. She has had two T & A operations, the removal of a lipoma from the back, and a ventral suspension. Is not deaf but has a sensation of closure and pressure in ears on awakening. Right sinus was operated eight years ago and still occasionally discharges. Records frequent attacks of pain in the heart with some dyspnea and palpitation. Presents a general history of gastrointestinal disturbance, some indigestion associated with the headaches, and chronic constipation. Menstrual history is negative. Physical examination shows a tendency to girdle obesity, with poor posture, heart normal, with some tenderness over the gall bladder and McBurney's point, small varicosities on legs, slight arthritic changes in hands and knees, but otherwise negative. Blood morphology shows pronounced lymphocytosis. Blood chemistry shows slightly high uric acid. The urine is negative except for a high residual nitrogen fraction. Serologicals are negative. Phthalein test is a low borderline. She is 14 per cent overweight; lung volume practically unimpaired. Basal rate is —14 per cent with a slightly lowered blood pressure and subnormal temperature. X-ray shows obscured antra, while the Barany is negative, as are the ears. The pelvic examination is negative. The neurological examination shows the patient somewhat slowed down and with a marked fine tremor of the hands. Her sugar tolerance is half the normal. Eyes show yellowish discs, enlarged blind spots, and a cutting of the upper fields due to lid droop. The endermal tests show sensitivity to several of the common foods. A duodenal function test (McClure) shows marked disturbance of liver function.

Diagnosis: The patient is not endocrine, gives clear-cut evidence of a liver dysfunction probably associated with gall bladder disease and of a questionable appendix.

Comment: Cases 9 and 10 are migraine, the focus showing as a dysfunctional state of the liver. An accurate history is always helpful and may indicate several foci as causative factors. The characteristics are that the unilateral pains are about twice as frequent as the bilateral; the former are rather more frequent on the left than on the right side, while it is frequently noted that the side varies in different attacks. The pain is, almost without exception, referred to the sinciput and deeply in the interior of the skull, never externally. Most frequently it is in the region above the eyes, in the center of the forehead, the temples, and the root of the nose. In severe cases it may extend to the upper jaw and the occiput, or even into the

nape of the neck. The degree of pain varies from trifling sensations to unbearable agony. Many patients are able, with an effort, to pursue their avocations, others become almost demented. The pain is most frequently described as boring or gnawing, and not uncommonly seems as though it would burst the skull asunder. All strong sense impressions, particularly bright light and noises, are appreciated as pain.

Summary: This paper seems to resolve into the fact that the nutrional level of the patient has a direct influence on the underlying increase or decrease of the symptom, headache. However, headache is seldom the result of a single focus, but rather is it the effect of several apparently dissociated conditions, *i.e.*, focal infections or intoxications in various parts of the body, especially the intestinal canal. On the other hand, the patent endocrine influence would seem to derive from their influence on general metabolism. The pituitary may be an exception to this; as certainly in cases presenting hyperplasia of the gland with a resultant intracranial pressure. Again, headaches in gonad disease are constant, are associated with menstruation and have an exacerbation period of added neuralgia, depression, and intensiveness usually cyclic in character. While the thyroid cases here cited are all liver dysfunction, it does not follow by any means that this is a constant factor. Finally, in a series of a thousand consecutive case histories that I have studied the order of frequency of headache as a presenting symptom correlates with the etiology as gonad, adrenal, pituitary, thyroid and pancreas, and that it is generally more frequent in hypo- than in hyperfunctional states of the ductless glandular system.

416 Marlboro Street.

SOME MOOTED POINTS IN SOUND CONDUCTION AND PERCEPTION DURING THE COURSE OF CHRONIC PROGRESSIVE DEAFNESS.*

DR. FRANCIS P. EMERSON, Boston.

In the following paper the writer will attempt to establish these propositions in relation to chronic progressive deafness:

1. That the conduction apparatus is never a factor during its course or in the end-result.

2. That it is an open question whether there is ever any obstruction in the Eustachian tube in chronic cases, except that due to trauma, tubercle or syphilis.

3. That nerve deafness is essentially like any other form of deafness.

4. That disuse never is followed by atrophy in a fully developed auditory organ.

5. That the only practical hearing test for otologists is the whispered voice.

In a discussion of the factors concerned in the problem of progressive deafness we can simplify the matter by leaving out of the question all those points over which there is no controversy.

Taking the conduction apparatus first, we will begin with the Eustachian tube. The infection of the glands at the mouth of the tube as part of the pathology in a tubotympanic catarrh is common observation. In cases of chronic tubotympanic catarrh we can see by the aid of a postnasal mirror or the pharyngoscope that the glands in the mouth of the tube have undergone a retrograde process, are atrophied and the shape of the opening so changed that it is now more triangular in appearance. Such tubes are easily entered with a cotton-tipped applicator or bougie, which can be readily passed up to the isthmus in advanced cases. When we remember that the tubal lymphatic tissue extends throughout the tube just beneath the ciliated epithelium, we can understand that the primary involvement of the lymphoid tissue in the fauces was followed by engorgement, hyperplasia, secondary atrophy and fibrosis within the tube itself. This brings us to the question of obstruction.

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During 35 years the writer has never seen but one tubal obstruction, except at the isthmus, and this seemed to be an annular ring near the isthmus. Many times there seemed to be obstruction present, but by changing the curve of the catheter they disappeared.

When we remember the angle that is formed by a line drawn through the tube as it meets the line through the floor of the nose, it is easy to see how an applicator or bougie would impinge on the wall of the tube and with increasing frequency as we approach the isthmus. In regard to narrowing at the isthmus, this is the one place that many believe is often obstructed. If we go back to our pathology we recall that there was first engorgement, swelling and hyperplasia, followed by atrophy. Now with this taking place in regular sequence, how can there be round cell infiltration and narrowing at the isthmus in a chronic salpingitis? Have we been mistaken? In approximately 900 dissections of the Eustachian tube, which were carefully made for teaching purposes at the Harvard Medical School, only two such obstructions were found either at the isthmus or at any part of the tube (Faunce). This again would be in keeping with our pathology, for an inflammatory process within the tube itself is never so virulent as to cause a break in the lining membrane, so that, when obstruction is found, is it not reasonable to suppose that it was caused by trauma, tubercle or syphilis? Clinically, obstructions of the tube raise the low limits in sound conduction, but this occurs during an acute tubotympanic catarrh, and as this clears up the low limits again return to within the normal range and are not again raised during the course of a chronic progressive case of deafness until perception for the whispered voice is nearly lost. Frequent obstructions of the Eustachian tube, then, are not probable from the pathology or clinical picture, either at the isthmus or at any other part of the tube, as the sequel to a low grade chronic catarrhal process.

Thickening of the membrana tympanum or infiltration, cicatricial tissue or bony ankylosis of the ossicles within the cavum impede or prevent the transmission of sound waves through the oval or round windows. These changes, however, occur so late in the course of chronic progressive deafness that they are an unimportant factor in the loss of tone perception. To bring out this point more clearly, let us trace the onset and course of all chronic cases of deafness. First, there is infection of the lymphoid tissue in the fauces, and with it tinnitus that is labyrinthine irritation. In many cases there may be hypersensitiveness to sound waves, so that patients wear cotton in their canals to tone down the painful sounds. There may

even be vertigo, with sensations of intratympanic pressure. The hearing is unnaturally acute and so far there is no involvement of the conduction apparatus, but every indication of irritation of the end organ.* In other cases tinnitus is present and patients say that it interferes with their hearing, but for some time there is but little change in the actual hearing test. If this process continues without involvement of the Eustachian tube and middle ear, the subsequent course is one characteristic of nerve deafness. If, however, there is an acute exacerbation or reinfection and a tubotympanic catarrh results, the low limits are raised and the middle limits are cut down. With the resolution of the tubotympanic catarrh the low limits become normal and the hearing throughout the scale may also return to normal. This process may repeat itself many times, leaving no indication of damage to the end organ, although there may be a ground-glass appearance of the membrana tympanum and thickening in the mucosa of the cavum. If, however, after one attack or several, the case is to develop into one of progressive deafness, the low limits return to within the normal range (this is stated advisedly, as the audiometer shows that the threshold of hearing is lowered throughout the scale), but the further progress of the case obtunds perception in the middle and upper part of the scale. In fact, the low notes remain normal until perception for the whispered voice is nearly lost in all chronic cases, even in so-called cases of nerve deafness. How can this be explained on the basis of a conduction deafness? During this time tinnitus is a common symptom, except in low grade cases of otitis media catarrhalis chronica, where there is lack of sufficient stimulation of the end organ, although here it promptly appears with each acute exacerbation. Patients complain that they cannot hear as well when tired. There is nerve depression and the hearing in bilateral cases tends to become equalized. The course is intermittent and not steadily progressive, as would be the case in increasing obstruction and immobility of the ossicles. Furthermore, with acute exacerbations of the local process that characterize its further progress, during the first 24 hours there will be tenderness over the tonsillar gland and hyperplasion on the side of the deaf ear, even though the patient may not be conscious of any local irritation in the fauces, in a large number of cases at all ages. As perception for the whispered voice is lost or lowered to from 1-25 to 4-25 the low limits for the first time are elevated. Now, what has caused the progressive loss in tone perception over all this time,

*The term "end organ" is used advisedly as the writer does not know whether we are dealing with a neuritis, perineuritis or ganglion infection. We do know that the more highly organized tissue is the most vulnerable and that the nerve is apt to be a secondary involvement.

which may cover a period of months or even years? We know that a tubotympanic catarrh undergoes resolution. Furthermore, we cannot conceive of a sticky exudate being present in the middle ear or tube without the low limits being raised, and yet after the acute attack they are not elevated until the deafness is far advanced. What is the etiology and what is to be gained by inflating an open tube with normal low limist? What is the cause of the elevation of the low limits at this stage? It was supposed to be due to an extension of the local infection in the fauces to the Eustachian tube, but it occurs in all cases, even in those of nerve deafness, at ust the same stage. Now, as the loss of tone perception is always a loss throughout the scale, it must be due to the threshold of hearing being lowered so that it now includes the low limits. This opinion is strengthened by the fact that when a case improves in tone perception, as occasionally one will if the hearing has been lost comparatively rapidly, the low limits improve first and there is no change in perception for the whispered voice until the low limits are again normal. In chronic cases we find, first, the whispered voice, the lowest note heard by air conduction, is lost or nearly lost with no obstruction in the middle ear or tube to raise the low limits up to this point. Further loss encroaches upon the low limit zone and they are then elevated. The bone conduction for a short time is increased and then lowered. There is never any variation from this orderly development in the gradual damage to the end organ in chronic cases, however we may classify them in reference to the changes in the middle ear. Following acute exacerbations of an infective process involving the tube and middle ear, and during its resolution, we may get any type of hearing test but when the case becomes chronic the loss of tone perception is registered in the same way in all cases, because it is a gradual clouding of tone perception which proceeds in an orderly fashion throughout its course. Lowered bone conduction with the raising of the low limits, then, is an indication of this regular, progressive course, and no new factor has been introduced either as an obstructive element or as a nerve involvement.

In suppurative cases the virulence of the primary infection usually involves the tube and does not clear up, resulting in an elevation of the low limits, but note that these low limits are not further elevated until tone perception is lost for the whispered voice. That is, following the acute explosion the further loss of tone perception follows the same course as in all chronic progressive cases, without regard to the pathology in the middle ear, until the hearing is irreparably damaged. So far, then, we have seen a local infection initiate a process that pursued its course as a case of nerve deafness. A similar

primary cause was followed by a low grade tubotympanic catarrh that underwent complete resolution, and then the chronic progressive deafness advanced in the same way as in the first case and ended in nerve deafness.

In the third type a virulent infection disorganized the middle ear and left an infected Eustachian tube, and after the case became chronic it ended in nerve deafness and showed the same development, in the same order, as in Types 1 and 2. At this stage no otologist could tell from the hearing test whether it was a case of deafness that had resulted from a chronic catarrhal or suppurative process, or from nerve deafness where the conduction apparatus was not a factor.

KEY TO HEARING TESTS.

W. V.—Whispered Voice.
Rinné—Determined by 256 C or 512 C^2.
Weber—Determined by 256 C or 512 C^2.
U. L.—Upper Limit, Monochord or Galton Whistle.
L. L.—Lower Limit, Dench Fork.

Case 1: Mrs. W. H. M. Age 56 years.
Head noises for years.
A. D. Eff. O. M. S.
A. S. O. M. C. C.

R.		L.
1/25	W. V.	½/25
18/13	Rinné 512 C^2	10/14
—	Weber	—
N	U. L.	N
128	L. L.	128

In the chronic stage they become alike without regard to the middle ear pathology in every case.

Case 2: Mr. McB. Age 28 years.
Duration, five years.
A. D. O. M. C. C.
A. S. Eff. O. M. S.

R.		L.
½/25	W. V.	½/25
18/13	Rinné 512 C^2	10/14
—	Weber	—
N	U. L.	N
128	L. L.	128

Different middle ear conditions, but the same tendency to become equal in the chronic stage.

Case 3: Mrs. J. Age 31 years.
Duration, five years.
B. Eff. O. M. S.

R.		L.
10/25	W. V.	10/25
70/45	Rinné 512 C^2	80/50
+	Weber	
N	U. L.	N
32	L. L.	32

Effect of a suppurative process but with the same tendency to become equal as the process becomes chronic.

It has been thought that a negative Rinné was evidence of middle ear deafness. No one disputes that the conduction apparatus physically impedes the passage of the low notes when changes have taken place, resulting in a thickening of the mucosa of the cavum or impairment of the mobility of the ossicles. A Rinné done with the 256 C^1 fork would be apt to give us a shortening in air and an increase in bone conduction, if there has been marked loss in tone perception, but this does not prove that the diminished tone perception was caused by the middle ear pathology and so far there has been no evidence that such is the case. Acute fixation of the stapes unquestionably results in marked deafness either by obstruction, increased pressure or as the result of the cause that produced it, but the further loss of tone perception as the case becomes chronic is characterized by the same course as that due to other causes. Where the stapes becomes fixed as the result of bony ankylosis, round cell infiltration or cicatricial tissue, the deafness is far advanced before this occurs.

On account of the nerve element and the inability to explain these cases on the basis of obstruction or conduction deafness there is a growing tendency to call all of them otosclerosis. Politzer, one of the best observers in otology, found but a limited number during his lifetime that could be confirmed by autopsy, and the number has not increased since that could be so verified. It is claimed that we may get any type of hearing test in otosclerosis, depending on the location of the sclerosis. This is true, but no case of infective deafness, however far advanced, ever showed a hearing test that resembled that of a typical case of otosclerosis. Moreover, the hearing tests in progressive deafness that result from a chronic infection are all consistently alike, without regard to the pathology in the middle ear or the type of infection. In otosclerosis no treatment influences its steady progress. It may have long intervals of quiescence, but it does not improve. What is the picture in infective deafness?

Treatment of the infected lymphoid tissue without any attempt to mobilize the ossicles has helped the hearing in hundreds of cases. That treatment of the nasopharynx improves the blood and lymph circulation and indirectly helps the middle ear is true, but such improvement often takes place following the removal of chronically infected tonsils without any local treatment. Also, the improvement in tone perception has been maintained until a subsequent reinfection has been followed by an increase in the deafness. Sometimes the good hearing has extended over one or two years without increasing and then suddenly has become worse during an epidemic of grippe or from an acute exacerbation of their local process.

Case 4: Mr. J. H. L.
Operated upon Jan. 29, 1923
A. D. Eff. O. M. S.
A. S. O. M. C. C.
March 7, 1923.

R.		L.
½/25	W. V.	1/25
7/0	Rinné 512 C^2	11/0
	Weber	+
N	U. L.	N
32	L. L.	32

Case 5: Sept. 10, 1923.

R.		L.
1/25	W. V.	25/25
12/4	Rinné 512 C^2	21/0
	Weber	+
N	U. L.	N
32	L. L.	32

Seven years before an instructor and a professor of otology had told the patient that he had nerve deafness and nothing could be done for him. He was very toxic from a recent exacerbation of a dental and antrum infection.

Case 6: Jan. 15, 1924.

R.		L.
1/25	W. V.	25/25
11/9	Rinné 512 C^2	26/11
—	Weber	—
N	U. L.	N
32	L. L.	32

Treatment: Abscessed second bicuspid that connected with the antrum was extracted, a radical antrum operation performed and a tonsillectomy. In September, 1927, he had a bad infection after going four years with good hearing.

Case 7: Oct. 17, 1924.

R.		L.
½/25	W. V.	25/25
21/11	Rinné 512 C^2	40/15
—	Weber	—
N	U. L.	N
32	L. L.	32

Right ear: Duration since childhood.

Left ear: Duration six months.

Case 8: Nov. 8, 1927.

0/25	W. V.	½/25
15/35	Rinné 512 C^2	40/31
—	Weber	—
N	U. L.	N
32	L. L.	32

Case 9: Mr. J. F. W.
B. O. M. C. C.
Dec. 2, 1911.

Age 51.

R.		L.
9/25	W. V.	16/25
N	U. L.	N.
32	L. L.	32

Case 10: March 15, 1913.

R.		L.
15/25	W. V.	7/25
N	U. L.	N
32	L. L.	32

Case 11: March 12, 1915.

R.		L.
12/25	W. V.	N
30/10	Rinné 512 C^2	35/13
—	Weber	—
N	U. L.	N
32	L. L.	32

This patient was a fruit broker who was often confused in buying at auction and would find he had bought two carloads instead of one.

Case 12: May 13, 1916.

R.		L.
Normal		Normal

Examination showed that he had a double chronic maxillary sinusitis, double suppurative ethmoiditis and sphenoiditis, and chronic tonsillitis. He has been operated upon for all these conditions. Previous to April 21, 1927, he had an acute infection. He is now 67 and after 16 years he is still buying at auction and has no trouble with his hearing.

Case 13: April 4, 1921.

R.		L.
3/25	W. V.	4/25
32	L. L.	32

Case 14: Feb. 8, 1923.

R.		L.
8/25	W. V.	7/25
47/17	Rinné 512 C^2	46/15
—	Weber	—
N	U. L.	N
32	L. L.	32

Case 15: Dec. 6, 1927.

R.		L.
12/25	W. V.	10/25
33/13	Rinné 512 C^2	32/11
—	Weber	—
N	U. L.	N.
32	L. L.	32

Case 16: R. R.
A. D. Eff. O. M. S.
A. S. O. M. C. C.
Jan. 9, 1922.

R.		L.
"Shout"	W. V.	2/25
17/30	Rinné 512 C^2	19/30
—	Weber	—
N	U. L.	N
256 C	L. L.	96 A

Case 17: May 29, 1923.

R.		L.
0/25	W. V.	25/25
14/41	Rinné 512 C^2	39/33
—	Weber	—
N	U. L.	N
64 C	L. L.	32 C

Right ear damaged in childhood. Left ear followed a submucous resection that went septic. When first seen there was a subacute nasopharyngitis. The patient was in the oil business and working on an alternating day and night shift, with bad ventilation and irritating chemical fumes. In October, 1926, he had a bad infection.

Case 18: Nov. 23, 1926.

R.		L.
0/25	W. V.	6/25
7/50	Rinné 512 C^2	75/50
—	Weber	—
N	U. L.	N
512	L. L.	32

Case 19: Nov. 30, 1927.

R.		L.
¼/25	W. V.	10/25
18/55	Rinné 512 C^2	63/70
—	Weber	—
N	U. L.	N
128	L. L.	32

Other cases with a fair amount of hearing, and therefore receiving a fair amount of stimulation of the end organ, will become suddenly worse and show signs of nerve deafness if they suffer from some general debilitating disease that not only lowers their resistance but adds to the toxic or bacterial damage to the perception apparatus. Furthermore, we cannot interpret perception damage in terms of merchanical obstruction, neither can we by the use of bougies, cotton-tipped applicators or inflation ever restore one note to the hearing acuity of a chronic progressive case of deafness.

Dr. Wilhelm Meyer, in 1860, made the epochal discovery of the relation of adenoid growths to deafness. The surgical removal of obstructing or infected lymphoid tissue in the vault of the pharynx has done more in the prevention and cure of deafness than all other measures combined. Not because it prevented or cured middle ear deafness, but because it prevented or cured perception deafness. As the result of these lymphoid masses in the epipharynx the tube becomes blocked and there is negative pressure followed by a passive congestion and effusion in the middle ear. This is, however, but of very little consequence in its relation to deafness, for the passive congestion undergoes resolution, leaving those notes which are affected by obstruction in the middle ear or tube normal. The infection, however, in the adenoid mass that was attended by loss of tone

perception in the middle and upper part of the scale was of vital importance to the future integrity of the end organ and must be cured of the infection of the lymphoid tissue and not by blowing out the tube on the theory of middle ear obstruction. Politzerization or inflation relieves a temporary condition that has nothing to do with chronic progressive deafness. It is questionable whether we ever have an active infection of any considerable mass of lymphoid tissue in the fauces that all of such tissue in this location is not also involved. The infiltrating tendency of certain strains of bacteria, together with a common lymph and blood supply, make this tissue which is undergoing retrograde changes exceedingly vulnerable. Then it depends on how far round cell infiltration, increased connective tissue with the formation of new blood vessels and the walling off of some pyogenic focus, has gone as to whether the local infection clears up at all. If not, increased tension and recurring discharge into the lymphatics may go on indefinitely, with a toxemia or bacteremia manifest at some distant point.

The evidence, then, points to infection as the starting point. They are made worse by infection that is acute or of low grade, acting through the lymphatics, accompanied by labyrinthine irritation, and they all end in nerve deafness. Whether the infection is of low grade or virulent, and whether the damage to the middle ear is gradual and results in round cell infiltration or is destructive to the conduction apparatus, the subsequent chronic course is one of progressive damage to the end organ. Where nerve deafness is demonstrable by the accepted hearing tests various theories have been advanced to explain its etiology. The most common have been atrophy from disuse, constitutional causes, rheumatism, disturbance in calcium metabolism, endarteritis, secondary impairment of the labyrinthine circulation leading to atrophy, chronic toxemia, etc.

Let us consider the first, atrophy from disuse. If we study analogous organs and see what happens under similar conditions, it may help us in answering this question. When the visual organ is undeveloped, if we cover one eye so that all light is excluded for two or three months, on removing the pad the patient is unable to see. If, however, we now exclude the light from the other eye the vision gradually returns from the stimulating effect of the light rays. On the other hand, if the visual organ is fully developed, covering one eye for the same length of time does not result in impaired vision on removing the pad. In the case of the auditory organ there is never at any time complete loss of stimulation of the auditory

nerve by the exclusion of sound waves by either air or bone conduction. What about senile nerve atrophy? Some five years ago the writer visited a home for aged women and examined a large number, whose ages ranged from 67 to 93 years. In all of them the relation of air and bone conduction was normal, even at 93 years, unless the case had been one of slow development similar to those under discussion. The writer has never seen a case of slowly progressive loss of hearing, from degenerative changes due to senility, that in any way resembled those in question. A certain amount of atrophy might, of course, take place in a nerve without any discoverable cause, except extreme old age, but the onset would be very late and not over a long term of years.

Constitutional Causes: This cause is very indefinite for a disease that afflicts such a large proportion of the population if applied to deafness. Metabolic changes that affect the blood cells or disfunction of the endocrine glands presupposes some primary cause back of these changes or disfunction. Depressed bodily states can lower the threshold of hearing but cannot initiate deafness. Chemical or metabolic changes by the generation of toxic products are undoubtedly important factors in the loss of tone perception, and are often secondary to the chronic infection in the lymphoid tissue, but in the large class where such changes do not exist it must be considered as playing a secondary role in the etiology of deafness. Constitutional causes that are followed by bony tissue changes surrounding the end organ of the auditory nerve we, of course, know nothing about.

Changes in the Blood Calcium: In 100 cases at the Evans Memorial Hospital there was no higher incidence in the blood calcium in patients with deafness than in 100 normal patients, and in both cases it approximated 5 per cent. This precentage is for the entire blood calcium, as we have no accurate test for the different calcium salts (Lawrence). Rheumatism, endarteritis and changes in the circulation of the labyrinth might all be the result of a toxemia or bacteremia.

Many arguments could be advanced pointing to the ganglion rather than the auditory nerve being at fault in cases of slowly progressive deafness. Whenever nerve atrophy as a primary or secondary result has set in, no treatment seems to influence its progress. That changes in the auditory brain center take place after a time seems probable from the fact that all chronic bilateral deaf cases tend to have a hearing test that is equal. Disuse and senility cannot be shown to be the cause and there remains only trauma and infection. The first

can be ruled out and we have only infection left to explain the etiology.

To sum up, then, we have a local infection in the lymphoid tissue that initiates the deafness, accompanied at once by tinnitus and sometimes vertigo and nerve depression. Tinnitus appears at intervals throughout its course and almost always with any exacerbation of the local process, the exacerbation being further connected with the increased loss of tone perception by tenderness of the sentinel gland and hyperplasia on the side of the deaf ear. The course is intermittent and influenced by treatment of the infected lymphoid tissue and never by any measures to mobilize the ossicles in chronic cases. The hearing test is typical after the disease is established and chronic, and does not vary from that of nerve deafness where there is no pathology in the middle ear. It never resembles a typical case of otosclerosis and never varies from its own characteristic development in slowly progressive cases. The end result in all cases is nerve deafness, which is made worse by any intercurrent disease that lowers the resistance of the patient, or by any local or general infection. If any of these cases improve by treating the infected lymphoid tissue alone, without any measures directed to the Eustachian tube or middle ear, they cannot be cases of otosclerosis or even of conduction deafness. While treatment of the nasopharynx influences the lymph and blood circulation in the tympanum, the same improvement in tone perception often follows the removal of chronically infected tonsils without local treatment. It does not make the observation of less value because they relapse, but it is highly significant that the improved hearing remains good for an indefinite time and then is lost through infection. Any obstruction in the middle ear does not modify the typical hearing test of a chronic case of deafness, as the middle ear pathology follows the loss of tone perception.

520 Commonwealth Avenue.

NASAL RECONSTRUCTION BY MEANS OF AUTO AND ISOGRAFTS.*

DR. MYRON METZENBAUM, Cleveland.

Nasal deformity cases requiring plastic operations for their correction from a practical surgical standpoint may be considered in two classes.

1. Cases where the deformity may be corrected by straightening, reshaping and reassembling the nasal bones, the cartilage and the soft tissues.

2. Cases where the loss of bone, cartilage or soft tissue may be restored partially or totally only by a transplant. In autografting, the tissues may be taken from the existing nasal structures or the graft may be taken from some other part of the body. In isografting the tissues may be taken from some other person whose blood is of the same type.

Case 1 was a malformation of the nose, face, upper jaw and lower jaw in a girl age 17 years. The lower jaw is very much retracted or underdeveloped. The upper jaw is protruding. There is a high or gothic-arched palate. The bones entering into the formation of the nose were out of alignment; namely, there was a dislocation of the perpendicular plate of the vomer and the septal cartilage at their junction with the perpendicular plate of the ethmoid. There was a dislocation of the upper border of the triangular cartilages with the lower ends of the nasal bones forming part of the large nasal hump. This hump was made up in part of the nasal bones, the upper portion of both triangular cartilages and organized callous. Through an internasal incision, the hump was carefully removed in its entirety and was transferred immediately to the nose of a young man who had suffered a nearly complete loss of the nasal septal cartilage. In addition to removing the hump, a V-shaped piece was excised from the end of the septal cartilage in order to shorten the nose. By chiseling off the premaxillary or premalar ridge, the upper lip was lowered so the mouth could be closed more easily. The septum was straightened and patency established to both nares. The photographs and plaster casts taken before and after the operation illustrate the improvement.

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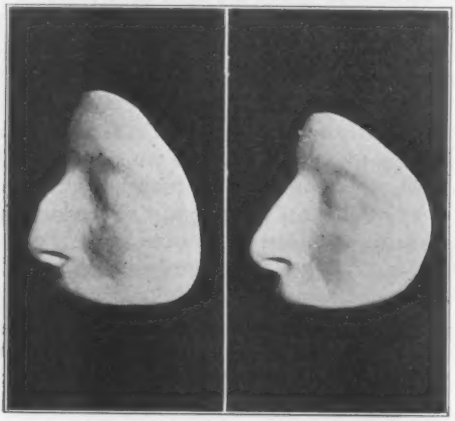
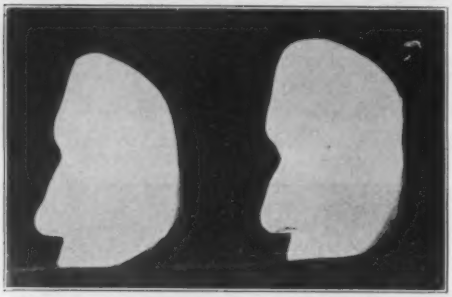
Case 2: Boy, age 18 years. One year previous to the operation, he was struck on the nose with a baseball bat. A hematoma formed between the nasal mucous membranes which suppurated and resulted in a nearly total loss of the cartilaginous septum. The nasal tip drooped and could easily be depressed down to the upper lip. This

Nasal hump with high gothic palate. Lower jaw underdeveloped. The hump was removed and transplanted into the nose of Case 2 to replace loss of cartilage.



Cast of Case 1 before operation. Cast of Case 1 after operation.

case could have been corrected by a cartilaginous transplant taken from the patient's rib, or a bone transplant taken from the tibia or possibly a fat and facia transplant taken from the thigh or an isograft taken from another person whose blood typed with his own. In this case the isograft was the *large hump removed from*



Cast of Case 2 before operation. Cast of Case 2 after operation.
Nasal tip raised by transplanting into the nose the LARGE HUMP removed from Case 1.

X-ray of Case 2 after insertion of transplant.
X-ray of the nose showing the transplanted hump taken from Case 1. The hump is lying downwards, THE SMOOTH or cut surface forming the straight bridge.

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Case 1. The X-ray picture shows the transplant inserted in the nose to replace the lost septal cartilage. The straight-cut surface lying upwards, forming the nasal ridge and the nasal hump, is downwards, filling in between the mucous membranes.

Isotransplants: Isotransplants of bone, cartilage, the small Tiersch grafts and the large Wolf grafts often fail to take, probably for the

Case 3. Front view before operation. Case 3. Front view after operation.
The large hump over the nasal bridge was removed and pushed downwards into a channel made in the fleshy part of the nose. This filled in the depression, raised the tip and re-established nasal breathing.

Case 3a. Side view before operation. Case 3a. Side view after operation.
The large hump over the nasal bridge was removed and pushed downwards into a channel made in the fleshy part of the nose. This filled in the depression, raised the tip and re-established nasal breathing.

same reason that many of the original blood transfusions failed. After the different types of blood were identified, it was found that only bloods of the same type are interchangeable and mix. When the blood of the donor is of the same type as that of the recipient, the transfusing or grafting of blood is successful in most cases. In patients whose blood types are the same, the isografting of blood, skin, fat, facia, bone and cartilage should give good results.

Case 1 belongs to the universal blood type No. 4, therefore she was a universal donor. Theoretically, her blood or tissues would serve as a transplant for any patient. Her Wassermann was negative. For these reasons I did not operate her when she first consulted me about her nose, but saved her for such a time until I should have a suitable case where I could transplant the large nasal hump made up of bone, cartilage and callous, into the nose of a patient requiring such a transplant.

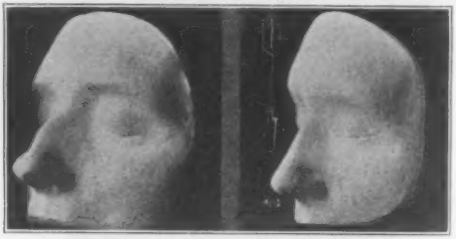
Case 4. Nose after insertion of FAT TRANSPLANT taken from patient's abdomen. The tip of the nose drooped, due to nearly a total loss of the septal cartilage and a perforation near the size of the lost cartilage.

Under local anesthesia the large hump was removed, wrapped in gauze and laid aside until the completion of her operation. The boy's nose was then injected with 2 per cent novocain and an incision was made through the skin along the upper portion of the tip of his nose. Through this incision a groove was made up to the end of the nasal bones and the upper portion of the mucous membrane. The ends of both nasal bones were freshened and the transplant inserted, with the hump downwards between the nasal mucous membranes, and the cut or the smooth surface upwards, to form a straight bridge, and the ends of the graft in contact with the freshened ends

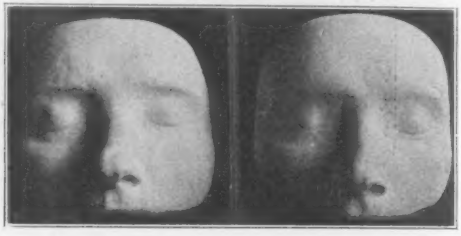
of the nasal bones. The incision was closed. The procedure for inserting the transplant was the same as is used in transplanting cartilage or bone.

Since this transplant is made up of bone, callous and somewhat thickened cartilage, the X-ray shows the transplant in place with the ends of the nasal bones, the hump is downwards, filling in between the mucous membranes, the straight surface above forming part of the nasal bridge.

The transplant lifted the nose, gave it good form and restored the nasal breathing and after one year and a half shows no absorption.



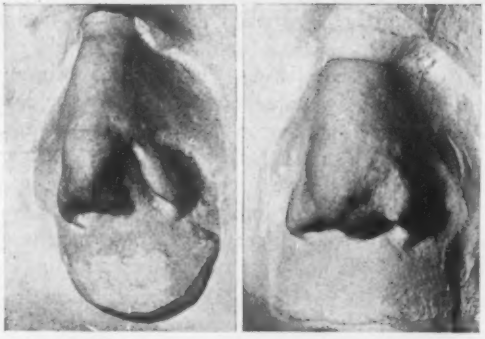
Left alar cartilage replaced by deflecting attached U-shaped piece of the septal cartilage into a channel made in the tissues of the left ala.
Before operation. After operation.



Case 5. Photograph of the front view before operation. Photograph of the plaster cast, front view, after operation.

Transplants—Shifting Tissues: Where a sufficiently large piece of cartilage, bone or callous or a combination of these structures exist in one part of the nose and can be shifted to another part of the nose to where there is a loss of similar tissue, the transplant usually takes. A common example is the transferring of a piece of cartilage from a lower lateral cartilage or from the septal cartilage and using it for filling in small depressions along the nasal bridge or to stiffen the ala.

Case 3: Due to a severe injury, the nasal tip was lying close to his upper lip. There is a very large perforation through the mucous membranes. A very large callous existed over the end of the nasal bones, which was used as an *autotransplant.* Through an internasal incision, the large hump was removed and pushed downwards into a groove made in the fleshy part of the nose and anchored in this position. The shifting of the callous raised the tip of the nose and



Case 6 Before Operation. Case 6 After Operation.
Ala restored by INTRANASAL FLAP. Tissues of the nose split intranasally, drawn forwards and free border sutured to septal cartilage.

is no reabsorption of the transplant. The front and side view photographs illustrate the changed condition.

Case 4: Following a septal operation there resulted a large perforation extending from the columella to the perpendicular plate of re-established the nasal breathing. After a period of two years there the ethmoid and from the floor to within ⅓-inch from the roof of the nose. The tip of the nose drooped close to the upper lip. In this case a simple cartilage or bone transplant was impossible, for

there was no septal mucous membrane in the nose to protect the transplant. The procedure to obtain the necessary lining to cover a cartilage or bony transplant for this part of the nasal bridge would have necessitated a great deal of intranasal surgery to form a mucous or skin lining to cover the cartilage or bony transplant, therefore a simple fat transplant was taken from the abdomen.

Under local anesthesia an incision was made through the tip of the nose and a groove made in the soft tissues along the dorsum of the nose. Then under local anesthesia a small mass of fat was removed from the abdominal wall and forced into the channel made along the dorsum of the nose and anchored by a through-and-through suture, with the result that the nose was raised up from the lip and given a dignified shape, the nares rendered patent and breathing re-established.

The photograph shows the result one year after the operation. Should the fat reabsorb partially or totally after a number of years, the reintroduction of more fat or facia through the original incision is rather a simple procedure.

Case 5: The photographs and casts show the nose fractured at the nasal bones and deflected toward the right. The dislocated inferior portion of the septal cartilage could be seen in the opening of the right naris. There was a partial loss of the alar cartilage of the left side, with a sunken in nasal wing. Through an incision along the inferior border of the dislocated cartilage, where the skin and the mucous membrane meet; the mucous membrane covering the cartilage was separated up to the nasal bones, the septal cartilage was then separated from the nasal bones, which permitted the cartilaginous portion of the nose to be swung into a straight line and also to replace the inferior border of the dislocated cartilage into its supporting position in the columella and thus stiffen the nasal tip.

Beginning close to the dorsal border of the septal cartilage, a small U-shaped incision was made downwards, leaving the cartilage still attached at its upper margin. The free portion was bent upwards and inserted into a channel made in the tissues of the left ala of the nose and anchored in place. This small deflected portion of cartilage which still remained attached along its upper border, substituted for the partially-lost alar cartilage, stiffened and obliterated the depression in the left ala, re-establishing patency to the left naris and made the tip of the nose appear alike on both sides.

Intranasal Flap: Case 6: Seventeen years prior to the operation, through an accident the man lost nearly the entire left wing of his nose. Due to exposure to the air, the tissues of the open nares were

thickened and the inferior and middle turbinates were atrophied to thin plates. The epithelium from the outside of the nose had grown over the borders and seemed to have replaced the normal nasal mucous membrane. The tissues were so thickened that an attempt was made to split the tissues from within outwards close to the edge and pull the inner split half outwards and attach its free border along the exposed septal ridge.

Leaving the upper border of the nasal wing untouched, the tissues within the nose were split from the floor as far back and as high up as the attachment of the nasal bone to the maxilla. The incision was then carried across the inside of the roof of the nose over to the septum. Then with a Freer angular knife, the tissues of the roof were split from behind forwards to within ⅛-inch of the edge. This entire dissected flap, which was still attached to the outer and upper free border, was pulled forward, bringing the mucous membrane surface outward. The one border was then sutured to a freshened area along the septal border to replace the missing tissue. The flap was slightly longer than necessary but contracted until it was somewhat too short. The flap also contracted laterally, slightly narrowing this nostril. The skin grew out from the nose, displacing the former mucous membrane. The denuded area within the nares became covered with what appeared to be mucous membrane. The picture shows the case six months after shrinking had taken place and shows the new ala as slightly shrunken and slightly collapsed. The operation was performed in 1918 and I observed the case for three years. The results are fully as good as could be obtained by taking a large sliding flap from the side of the nose or forehead and was not as disfiguring. I believe this to be the first time that the ala of the nose was restored by an intranasal flap. The shrinking of the flap can now be avoided by the method devised by Mr. Gillies, of London, namely, to provide a skin lining for the flap taken from the arm or thigh of the patient.

736 Rose Building.

CASE REPORT. OPEN SAFETY PIN REMAINING STATIONARY IN THE THORACIC ESOPHAGUS OF A BABY AFTER FIFTY MILES ROUGH TRANSPORTATION.*

DR. FREDERICK T. HILL, Waterville, Me.

We would generally consider that a foreign body which has passed below the cricopharyngeus constriction in the esophagus would go on into the stomach, provided the esophagus is normal. We would expect this to happen, especially in a child with a safety pin below the cricopharyngeus, who was transported 50 miles over a rough New England road in January. The retention of the pin in this location, after such a trip, with the keeper apparently held up, or hooked over the narrowing of the esophagus, due to the crossing of the left bronchus; and the subsequent dislodgment of the pin downward into the stomach incident to esophagoscopy, without its being seen, or apparently touched, is the excuse for the reporting of the following case.

Baby E. P., age 15 months; while changing the baby's clothes at 6:30 a. m., the mother dropped an open safety pin, 1 inch long, into the child's mouth. She made the usual futile attempt to recover it. There was some choking but no coughing, and the pin disappeared. The family physician was called and an X-ray was taken, showing the pin apparently in the esophagus behind the heart shadow. The doctor sent the child, together with the X-ray he had taken, to the Sisters Hospital at Waterville, a distance of about 50 miles. The child arrived at the hospital about 2 p. m. I saw the case about two hours later. Inasmuch as the trip had been made over rough roads, made worse by January weather, it seemed quite probable that the pin would have moved from its earlier position. However, X-rays taken after admission showed it to be in its original location, at the level of the fourth thoracic vertebra. The keeper of the pin was apparently just above what appeared to be the outline of the left main bronchus, where it crosses the esophagus. The point was up.

The child had not taken any nourishment all day, refusing to swallow, and, as a consequence, was not in good condition. Without anesthesia, after the larynx had been carefully inspected with a Jackson speculum, a small esophagoscope was introduced into the esophagus. There was a large amount of mucus, requiring almost constant use of suction. The pin was not seen, despite careful search.

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*From the Otolaryngolic Service, Sisters Hospital, Waterville, Me.

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The esophagoscope was immediately withdrawn and the procedure discontinued. The baby was returned to bed and a retention enema of coffee and bicarbonate of soda given. It was not considered wise to disturb the child for a second X-ray examination that evening. He reacted well and at 6:30 p. m. took three ounces of milk, the first food he had been able to take that day.

X-ray the following morning showed the pin evidently in the cecum. The case was transferred to the surgical service, under the care of Dr. A. H. McQuillan. The child was in much better condition and taking nourishment. Daily observations by means of the X-ray showed the pin progressing onward. One day later it was seen in the descending colon, and the day after in the sigmoid. That afternoon it was recovered from the stool. The following day the child was discharged home, recovered.

One would naturally expect a pin which had passed the narrowing of the esophagus at the level of the superior aperture of the thorax to pass on to the stomach, especially after the jolting incident to a 50-mile ride over winter roads in a small car of popular make. From careful study of the X-rays it seemed that the keep of the pin was held up by the crossing of the left bronchus, pressing on the esophagus. This could be made out quite definitely in the X-ray. Straightening of the esophagus incident to the passing of the esophagoscope must have released the keeper. As the head was in the "high position" until after the bronchus was passed, gravity would have aided the released pin to slip downward into the stomach. Possibly had the table been tilted with the head lowered, rather than horizontal, it might have been better. Of course there is the possibility that the pin was accidentally pushed downward into the stomach, although I do not really think this occurred. The pin was never either seen, or felt, nor was any forceps or other instrument inserted in the tube. The esophagoscope was passed downward just once, slowly and as carefully as possible with a baby naturally frightened and struggling.

That the procedure resulted in the passage of the pin to the stomach would seem indicated by the baby's taking nourishment shortly afterwards. Immediate X-ray would have been interesting, but the baby's poor condition, prior to endoscopy, with the added shock and fright incident to the procedure, short as it was, made me forego further investigation until later. The continued progress of the pin in the intestinal tract made operative interference unnecessary. This case would seem to bear out the wisdom of not prolonging any such procedure, even though failure to produce the foreign body for the edification of the operating-room audience may be humiliating, or seem to be, at the time.

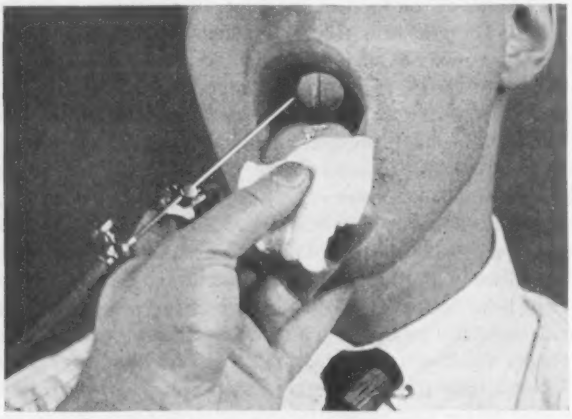
Professional Building.

FINGER HOLDER FOR LARYNGOSCOPIC MIRROR.

Dr. M. Joseph Mandelbaum, New York City.

Indirect laryngoscopy, despite the increasing use of the direct method, still occupies an important place in the diagnosis and treatment of laryngologic conditions. Many laryngologists are hampered in their work by their inability not so much to bring the laryngeal image into the visual field, as to keep it there for a long enough period to make a satisfactory diagnostic survey or a proper application of the indicated therapeutic measures.

Chief among the factors tending to interfere with the visualization of the image in the laryngeal mirror is the patient's tongue. During



the intralaryngeal instrumentation by the indirect method, either an assistant or the patient himself holds the tongue.

In either event, considerable difficulty is frequently experienced by the inability of a hand other than the specialist's to properly co-ordinate with the latter's hands or his line of vision.

Hence, any method to obviate such extra assistance, and at the same time permit all of the necessary manipulations to be performed and controlled by the laryngologist himself, is a welcome innovation.

Several years ago I devised a simple appliance which permits the operator to perform laryngoscopic instrumentation by the indirect

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method without assistance. It consists of a ring-like flat band made out of spring steel, not however forming a complete circle. The band may be compressed so as to fit fingers of varying degrees of thickness. Opposite to the cleft in the band on its outer convex surface is fastened a universal joint containing a short transverse canal, through which is passed the narrow shaft of an ordinary laryngeal mirror. Another small channel running vertically, at right angles to the horizontal canal with which it communicates, is threaded to receive a thumbscrew, by means of which the position of the mirror is fixed in any desired position.

This device permits the laryngologist to hold the patient's tongue between his thumb and middle finger, while the mirror holder, placed upon the first or second joint of the index finger of the same hand, holds the laryngeal mirror in position, thus giving the examiner absolute control of the patient's tongue, dispensing with the need of its being held by the patient himself or an assistant. The mirror holder is made to hold the ordinary laryngeal mirror shaft.

The appliance is made by Pilling and Company.

125 West 72nd Street.

FIRST INTERNATIONAL OTO-RHINO-LARYNGOLOGICAL CONGRESS.

Sailing from New York, July 6, 1928.

Eye, ear, nose and throat doctors of the world will meet for the first time at the First International Congress of the Oto-Rhino-Laryngological Society, to be held in Copenhagen, Denmark, July 29 to August 1. That was the announcement made by the American Committee of the Society, 25 Broadway, New York.

More than 75 specialists will represent the United States at the Congress. These doctors will also spend some time visiting at various large cities in France, England, Germany, Norway and Sweden. Clinical discussions will be held in these countries with European doctors presiding.

The Congress will concern itself with questions relating to the treatment of the many maladies, injuries and infections of the eye, ear, nose and throat. It has been reported from abroad that very successful methods have been found for sinus trouble and middle ear deafness.

A COMBINED FRONTAL SINUS DILATOR AND CANNULA.*

Dr. William Spielberg, New York City.

The frontal sinus dilator or sound is an instrument used in the intranasal operation on the frontal sinus. It is also employed in the diagnosis and postoperative treatment of diseases of the frontal sinuses.

Without the aid of these sounds, an intranasal frontal sinus operation would be more difficult to perform and its successful termination or cure made practically impossible. In the postoperative treatment

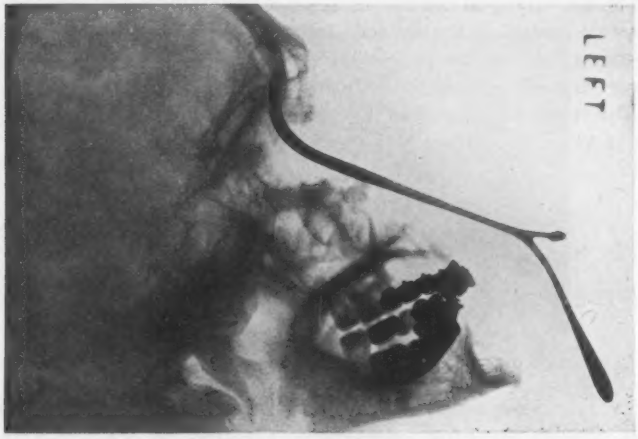

Fig. 2. Left. Case of postoperative chronic suppurative frontal sinusitis showing the dilator-cannula inserted into the left frontal sinus. X-ray plate taken by Dr. L. J. Friedman.

of chronic suppuration of the frontal sinus the continuous passing of the sounds into the sinus for a long period of time prevents the opening made into it from being closed by granulations or contracture of the surrounding tissues.

Due to the accumulation of secretions in a postoperative frontal sinus, it occasionally becomes necessary to irrigate it. In such instances the patient is subjected to two procedures, namely that of

*From the Otolaryngological Department of the Beth Israel Hospital.

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inserting the sound, then to be followed by a frontal sinus cannula. The latter procedure is usually very painful and causes considerable trauma, due to the nonconformity of the curve of the frontal sinus cannulae in use at the present time, with that of the nasofrontal duct or the opening made into the frontal sinus.

The curve of the frontal sinus dilators, however, conforms so well with both the normal nasofrontal duct and the opening made by the operation on the frontal sinus that we thought it might also be used as a cannula by having the sounds made of tubular construction, thus combining the dilator and cannula into one instrument.

The resulting instrument was found to be very desirable and advantageous, for the following reasons:

1. It spares the patient unnecessary pain and trauma as a result of too much manipulation, as compared to methods used heretofore.

2. This instrument conforms so perfectly to the curve of the nasofrontal duct or opening made into the frontal sinus postoperative, as to surpass in efficiency any other frontal sinus cannula made.

I am indebted to Mr. Stumer, through whose courtesy and co-operation the instrument is being manufactured by the Pfau American Instrument Co.

211 Henry Street.

ALUMNI ASSOCIATION MEETING, NEW YORK EYE AND EAR INFIRMARY.

Saturday, April 28, is the date decided upon by the executive committee for the next annual meeting and dinner of the Alumni Association of the New York Eye and Ear Infirmary. An attractive program has been arranged at the Infirmary during the morning and afternoon.

THE NEW YORK ACADEMY OF MEDICINE.

SECTION ON LARYNGOLOGY AND RHINOLOGY.

Regular Meeting, Nov. 23, 1927.

The Location of the Focus in Optic Nerve Disturbances from Infection.

Dr. Leon E. White (by invitation).

Some focus in the body is responsible for the optic nerve lesion arising from infection. Invasion is through the blood stream. The accessory sinuses supply but an insignificant number of the foci. Most of the foci are found in teeth and tonsils. The sinuses from their priority of investigation have occupied the attention of rhinologists too long to the exclusion of other more frequent seats of infection. Much can be learned from the general medical man on the best methods of investigating focal infection. Dental infections are easily overlooked. An expert odontologist is essential in a successful search for dental pathology. Devitalized teeth are a potential source of infection. Tonsils are a frequent source of infection and should be removed when at all suspicious if no other foci are found. The antrum among the sinuses is the most frequent seat of infection. Thickening of the antral mucosa without secretion may be a sufficient focus to warrant surgical intervention. Definite infection in the other sinuses should be eliminated, but this is of rare occurrence. Marked nasal blocking should be removed. Ventilation of the sphenoid as a therapeutic measure is rarely indicated but is still advocated when optic atrophy is imminent. The infection may be in remote regions of the body. The focus may be found in the appendix, gall bladder, prostate, Fallopian tubes, genitourinary or intestinal tracts. Systemic infection following influenza, scarlatina, etc., may be responsible for the optic nerve lesion. Local treatment is advocated in addition to the removal of the focus.

Normal vision was obtained in 6.3 per cent of the cases undergoing operations, while 20 per cent improved. In the remaining 17 per cent, which were chronic, further loss of vision was prevented. The ethmoids were opened but once, the sphenoid twice, and the antrum four times. Over 80 per cent of the cases had infected teeth or tonsils, or both.

DISCUSSION.

DR. COLMAN W. CUTLER: It is not easy to discuss Dr. White's paper, as there are many points which require careful consideration, and I find that the statistics which he cites leave a confused impression as to cause and effect of treatment. In much I must differ, and I do not think that his point that most of the cases of retrobulbar neuritis have been cured by removal of teeth or tonsils, is proven. It would have been interesting if some of those cases which recovered after such excisions had been treated simply by the use of the ordinary nasal douches and the frequent use of adrenalin or epinephrin, as is recommended by many observers. The fact, as I shall state later, that the early stage, at least, of retrobulbar disease is due to an intense edema makes the rapid recovery in many cases, after such apparently simple treatment, perfectly comprehensible. I am sure, however, that all of us are grateful for Dr. White's conservative views, and it is certainly conservative to remove diseased teeth and tonsils whether they are the actual cause of the optic nerve disease or not. It will relieve us, as ophthalmologists, of a grave burden of responsibility if we are convinced that the optic nerve is so rarely threatened by nasal obstruction and edema or infection through the posterior sinuses.

Teeth and tonsils have been in our minds for a long time, but the infection from these sources is more apt to cause disease of the iris, the choroid and the ciliary body, namely uveitis, less frequently of the retina and retinal vessels, and rarely of the optic nerve. I confess I have not seen such a case. In all cases of ocular inflammation, however, one thinks first of the teeth and tonsils and no conscientious specialist would attempt a diagnosis without all the aid of the internist and the resources of the laboratory and allied specialists.

We are discussing, however, that part of the optic nerve which lies within the bony walls of the optic canal, as that is the part, if any, which is involved

by its proximity to the posterior sinuses. To enter into the discussion of papilledema and orbital forms of optic neuritis which arise quite as often from systemic or bone disease, or other infections, would enlarge the field of this discussion unduly and, it seems to me, deprive it of its special value, which is to delimit the type of retrobulbar neuritis which ophthalmologists have recognized as especially associated with the posterior sinuses.

A recent article by Prof. Meller and Dr. Hirsch, of Vienna, "Ueber die Rhinogene Neuritis Retrobulbaris", is well reviewed by Dr. Knapp in the Archives of Ophthalmology, of September, 1927. The original article should be read by all who are interested in this subject. It is conservative in tone and contains some pathological evidence which supports the hypothesis of Van der Hoeve and which also duplicates the important work done by Herzog on the pathology of the sinuses and the mode of extension of the inflammation through the bone to the walls of the optic canal and the sheath of the nerve.

In this paper of Meller and Hirsch, as well as in one which I was permitted to read before this Section in 1922, clinical evidence was offered to show the existence of a true rhinogenic retrobulbar neuritis. The cases may not be numerous in proportion to the whole number of cases of nasal disease. They seem to occur more often at certain seasons or years, as if epidemic, as do other inflammations of nose and bronchi. This has been remarked by several writers. They are not always easy to detect, as a central loss of vision is not constant nor is it always an early symptom, and the ophthalmoscopic signs are slight. In fact, if we wait until there is pallor of the nerve, irreparable damage may be done.

The paracentral scotoma and enlargement of the blind spot have been sufficiently described in earlier papers.

Granting, as we must, that these cases do occur, may I ask my friends, the rhinologists, these questions:

Does the inspection of the nose suffice to exclude infection or disease of the accessory sinuses?

Is it possible in all cases, by straightening the septum and by shrinking the turbinals, to drain the ethmoid cells?

It has been stated repeatedly, and it is a point of much importance, observed by many of us who have been interested in these cases, and who have worked in close relationship with rhinologists, that the absence of secretion is not to be taken as excluding nasal disease which may cause rhinogenic retrobulbar neuritis. In fact, Hajek's statement, "Pathologisch-Anotomische Befunde fehlen so gut wie vollstandig", has seemed to make this problem more obscure. Indeed, in the minds of many it has been the strongest reason for doubting the association of nasal disease and retrobulbar neuritis.

In the light of this obstacle, the second part of the contribution of Meller and Hirsch, the rhinological part, by Dr. Oskar Hirsch, is more important than the ophthalmological part, which merely adds facts to those already known, as it bears out the statement of Herzog, in 1921, that the mucous membrane which was apparently not pathologically changed, showed, nevertheless, microscopically, inflammation which extended to and through the bony walls of the optic canal.

Herzog regards rhinogenic retrobulbar neuritis in most cases as the result of an inflammatory edema of the sheath of the optic nerve. If this view is accepted, it makes the fluctuations of the retrobulbar neuritis less obscure.

If the sheath of the nerve and its outer layers are involved, the clinical symptom is the enlargement of the blind spot. If the edema compresses the nerve, the delicate papillomacular bundle is affected and a central scotoma follows. If the nerve trunk is invaded, the field of vision suffers in various ways.

Edema may be relieved very promptly by adequate drainage. Hence the prompt recovery of vision following, in some cases, an early reduction of the nasal edema and obstruction.

Let me say in closing that, while I applaud Dr. White's thoroughness in studying teeth and tonsils, when the vision and the field show signs of retrobulbar neuritis, and especially if the changes in the field are progressive, one should not postpone treatment of the nose, the details of which it is not my place to tell you. Some mild cases may recover with little treatment, but that

is not a sufficient reason for directing attention to remote sources of infection and a problematic blood stream infection, unless or until adequate treatment of the nasal field has been begun. The outcome may leave one in doubt, as in so many medical and surgical puzzles as to where the chief burden of the cause lies.

DR. JOHN M. WHEELER: I wish I could say as gracefully, as courteously as Dr. Cutler has done that I cannot fall in line with Dr. White in his consideration of the disturbances of the optic nerve. I think we have launched forth on the discussion of a very important subject without first getting our bearings. If one were to judge only by Dr. White's remarks in regard to the etiology of optic nerve disturbances he would have a very wrong impression. Before considering the causes which Dr. White has mentioned, the laryngologist should have a general idea of the possible causes of optic nerve disturbances, so that he can keep his bearings. There are many possible causes of pathological changes in the optic nerve which have to be taken into consideration. Some few cases are due to traumatism, operative or nonoperative. Then there are the important cases of "primary optic atrophy" which may be due to a very slow-growing inflammatory process which results in blindness in both eyes—irreparable blindness. Bilateral papilledema is usually caused by expanding lesions in the brain, with increased intracranial pressure, and perhaps 70 per cent of these are neoplasms. Dr. White mentioned one neoplasm, inferring that they are rare cases. In fact, neoplasm is a common and very important cause. Another very important cause of optic nerve disturbance is hypophyseal enlargement. Many hypophyseal cases have had nasal operations, and it is a reflection on the profession that this is so. Hypophyseal enlargement is a common cause of atrophy of the optic nerve and this should be eliminated before the sinuses are operated on. Suprasellar tumors form another casual group. Not so long ago a nurse came from another city to New York and was made a supervisor in a hospital. She had had teeth and tonsils extracted and her nose operated on for an optic neuritis. She came to our hospital and we found pus in the sphenoid; pus was washed out again and again, and we thought we had optic neuritis, due to invasion of the accessory sinuses, but later a large neoplasm was found in the vicinity of the optic nerve. We should have known better, and have known right away that this was a case of intracranial lesion, and if we had studied that patient and her fields of vision intelligently enough, we should have made a prompt instead of a delayed diagnosis. Syphilis is a common cause of optic neuritis, sometimes associated with papilledema and sometimes not. Aneurysm or rupture of the internal carotid within the cavernous sinus is another cause of optic nerve involvement. Encephalitis and meningitis should not be forgotten as possible causes.

Dr. White properly spoke of a few febrile conditions which may occasionally cause optic nerve affections. Then there is myelitis. I saw a patient recently who had lost the sight of one eye and the other was affected. A diagnosis of retrobulbar neuritis was made and a neurologist ordered a nose surgeon to operate on the sphenoid sinuses and the patient's sight improved; but it was not long before it was found that it was a case of myelitis, and paralysis set in. It would have been very nice if the nose surgeon had known of the possibility of myelitis in this case and had refused to operate. For blindness may be the first manifestation of myelitis.

Alcohol and tobacco may cause an optic neuritis with amblyopia, especially in cases with loss of central vision in each eye. The possibility of injury from other poisons also has to be considered.

Without doubt inflammatory processes in the ethmoidal and sphenoidal sinuses, whether purulent or nonpurulent, may occasion optic neuritis. Dr. Cutler has already referred to the pathology in such cases. An edema may invaded the optic nerve sheath as a result of sphenoiditis and ethmoiditis. Very rarely a similar process might result from antrum disease. It is possible that an abscess at the root of a tooth may cause a papilledema and a retrobulbar process. It is just as possible that infected tonsils may cause an optic neuritis, although I have never been convinced of that etiology in any case. It is barely possible that infections of the appendix, gall bladder, male genitourinary tract,

of female genital system may cause an optic neuritis. I doubt whether they have, but it is possible.

Let us say that the services of the rhinologist may be very important in a relatively small proportion of optic nerve disturbances (I wish we were discussing a definitely conceived lesion of the optic nerve) on account of the anatomic arrangement that you are familiar with. But let us say that remote foci, such as Dr. White has mentioned, should occupy remote places in our mental processes when we consider etiology of optic nerve disturbances.

DR. LEWIS A. COFFIN: Before entering into a discussion of Dr. White's paper I would like to call attention to that condition of the optic nerve produced by brain tumor giving rise to choked disc in which the nerve head may be swollen to the extent of 7 or 8 diopters—a papilledema. The nerve fibres are separated from each other by cerebrospinal fluid. Please get the picture well in mind, it is not an "itis" and is concededly due to pressure which interferes with the return flow of blood and cerebrospinal fluid.

Now a condition of the optic nerve not infrequently arises from sinus disease which so simulates the condition produced by brain tumor that no man can objectively distinguish between the two. This condition arising from a diseased sinus is also produced by a retarded return flow of the fluids of the parts, due to the phlebitis condition of the ophthalmic veins. In the brain tumor cases the return flow is retarded by pressure on the veins. In the condition arising from sinus disease the return flow is retarded by a diminution in the calibre of the veins. I have pictures of these pathological findings to prove this point. In my opinion it is impossible that this condition of the nerve arising either from brain tumor or phlebitic ophthalmic veins can be produced by any blood-borne virus, toxin or organism capable of affecting the optic nerve. The edematous condition described as arising from brain pressure or phlebitic ophthalmic veins is probably not properly designated optic neuritis; whereas that affection of the nerve from a blood-borne virus is a proper neuritis. Both conditions may lead to nerve atrophy.

Subjectively, there is this marked difference. A person whose nerve is affected from brain tumor or from sinus disease as above described may continue for some time to have practically normal vision, whereas one of the first symptoms complained of by a person whose nerve has been affected from blood-borne toxin is that he is suffering from blurred vision.

Malignant pustule on the lip produces the same condition of the optic nerve as has been described as arising from the pressure due to a brain tumor or a phlebitis of the ophthalmic vein because the veins supplying the lip empty, as do the nasal veins, into the ophthalmic. A malignant pustule on the hand will not produce this condition. As Dr. White has said, many of these changes in the optic nerve, aside from those arising from brain tumor, tend to recovery and anything that we may do that has a tendency to improve the circulation about the nerve will increase this tendency to recovery. The removal of the tonsils has a marked tendency to open up the occluded nostril, which improves the circulation.

I have no quarrel with Dr. White as to the ability of a focal infection anywhere in the body to produce an optic neuritis, and I can even conceive of an optic neuritis caused by blood-borne toxin from a diseased sinus, but it is not the condition of the optic nerve usually produced from a diseased sinus. In other words, I believe that we may have either a papilledema or a proper neuritis from sinus disease, but I must differ with Dr. White as to the relative frequency of the nerve disturbances arising from sinus disease, as compared with those arising from foci in more distant points of the body.

DR. MARTIN COHEN: I can add nothing except to agree with some of the speakers in regard to the histological, anatomical and pathological condition. I don't see how it is possible for a tooth infection or a tonsil infection to produce any optic nerve lesions; it is entirely out of my line of thought. It does not seem possible.

DR. BEAMAN DOUGLASS: I am pleased to be able to add my word of praise and thanks to Dr. White for giving us this paper. It is now more than several years since he began to work so industriously and so assidiously on this subject. Tonight he has shifted his position entirely from that expressed in his early paper. I admire his frankness in that change of view; it is the

result of honest conviction, and I think with Dr. Coffin that Dr. White is progressing.

One might discuss this paper from the many points of view that Dr. White presented tonight, but such discussion would be interminable. I shall confine my remarks to referring to something very lightly touched upon by the Doctor in one part of his paper. When one sees a case of optic neuritis and frank sinus involvement, and the sinuses are opened and pus is found, and then two or three days afterward the patient has recovered his sight—such a result is very remarkable and very desirable, and today thoroughly understandable. When, on the other hand, a patient presents himself with an optic neuritis and the sinuses are opened and found normal, and the mucous membrane lining the sinuses is normal, and still that patient improves, and in two or three days or a week his blindness has disappeared—such a result is more remarkable, just as desirable, but not so understandable; and, lastly, when such a patient goes from the ward to the operating room with a frank diagnosis by a competent oculist of an optic neuritis, and when that patient is practically blind, and on returning from the operating room, having had nothing done but a middle turbinate snared or a submucous operation, and when returning to the ward that patient is able to read a watch immediately—such a case is very miraculous, very desirable, but entirely incomprehensible unless we refer it to those fertile fields of hysteria in which grow such extraordinary results.

DR. DWYER: This is a very timely paper. In my opinion, however, a lot of the ambiguity and contradictions regarding such cases is due to the fact that the nose and throat men do not appreciate the viewpoint of the eye man, and vice versa. From the standpoint of the nose and throat, I wish to assure Dr. White that it is not the opinion of the New York men that most of the focal infections come from the sinuses; on the contrary, I think very few do. Again, the optic nerve lesions from the sinuses more often come by direct pressure or extension than by other means. From the eye standpoint, there is a certain amount of contradiction in the various terms used in describing what is probably, or may be, phases of the same pathological process or different processes. Thus, the terms optic neuritis, choked disc, papillary edema and retrobulbar neuritis are used rather loosely as to their meaning, as we look over the literature. I think that edema of the optic nerve from pressure or from an adjacent or contiguous infection ought to be sharply differentiated from a true optic neuritis. They are different processes entirely, and have been shown to be so in his experience. The whole question is still very much sub judice, and we ought to proceed very slowly in drawing any radical conclusions.

DR. WHITE (closing the discussion): When Dr. Cutler mentioned that dental infection in eye disturbances was no new idea but one that had frequently been called to the attention of the ophthalmologists, I began to feel that I had been telling you about infection with which you were so familiar that this plea for a more thorough investigation for other foci before resorting to haphazard surgery on the accessory sinuses, was entirely out of place.

As the discussion progressed, however, I discovered, as I had suspected, that the accessory sinuses were still paramount in the minds of most of you, so that I feel that after all it may have been worth while to me to come here, especially if my efforts succeed in diverting your minds somewhat away from the sinuses. The sinuses, of course, do sometimes harbor infections that are capable of producing optic neuritis. Their importance has, however, been vastly overestimated. I have endeavored in this paper to place them in the background where they belong. In doing this I realize that I have belittled them in the minds of most of you, so I am not surprised to find them so ably defended.

At the summer graduate course in ophthalmology and oto-laryngology in Denver this past season these borderline optic nerve cases were discussed by Drs. H. L. Baum, W. I. Lillie and myself, and although I did not hear much of the discussion I was told by one of the students that we all agreed that accessory sinus surgery had been greatly overdone and that the infection was most commonly found in teeth and tonsils.

Dr. Cutler asked the rhinologists present if one was able to determine nasal infection by simply inspecting the nose? Let me say that most assuredly it

cannot usually be so easily located. This phase was touched on but briefly in the paper. I did say that, "If transillumination and radiographs confirm my findings on inspecting the nasal cavities and these are negative, I immediately look elsewhere for a focus."

Dr. Cutler has quoted several authorities (mostly German) in support of the accessory sinuses. Most of these were five or more years ago. At that time I suspect he might have found support from the papers I had published principally stressing the accessory sinuses. The paramount importance of dental and tonsillar infection is very recent. At a joint meeting of the Royal Society of Medicine between the Section of Ophthalmology and Laryngology during the past year I was gratified to see that the tide had turned and that focal infection from teeth and tonsils was receiving the attention it merited. The consensus of opinion was, according to Dr. Campbell Smyth, who reviewed the papers presented in this meeting in the September, 1927, Archives of Oto-Laryngology, that "inestimating the beneficial effect on the vision of surgical intervention in the cavities of the nasal or posterior sinuses due regard must be paid to the fact that in cases of retrobulbar neuritis there is a tendency to spontaneous cure, and the operation may be performed at a time when the vision is commencing to undergo natural resolution. The necessity of cleaning up obvious foci of infection in the teeth, tonsils or any other part of the body was emphasized."

Dr. Coffin misquoted me a little in stating that I said in my previous paper before this Academy that I "had no use for suction". The statement was that "I had not used suction in these cases." He is also mixed on the ethmoid case. I stated that in the case with pansinusitis I only removed the anterior ethmoids so as to facilitate drainage of the frontal. The pansinusitis was acute and I did not consider it at all necessary to open the posterior ethmoids and sphenoid The rapid recovery proved that enough had been done.

In reply to Dr. Wheeler let me remind him that this paper only deals with optic nerve disturbances from infection, and naturally no attempt was made to consider retrobulbar neuritis or papilledema due to alcohol, lead, tobacco or intracranial neoplasms. Before writing this paper I discussed this title with one of the eye men as I wished to limit it largely to infection such as I had frequently found in teeth and tonsils. Let me say in passing that the so-called papilledema arising from infections in teeth, tonsils or sinuses occurs but rarely. In my series but 4 per cent have shown swelling of the disc. This was unilateral and associated with marked loss of vision, while true papilledema due to intracranial pressure is usually bilateral and associated with little or no loss of vision. Papilledema, therefore, usually arising from intracranial pressure should direct our attention to the brain and I always refer these casese to a neurologist.

Only after the exclusion of intracranial conditions would one be justified to submit these cases to nasal surgery. At the meeting of the Academy of Ophthalmology and Oto-Laryngology in Montreal three or four years ago, I took part in a symposium on papilledema and advocated the following change in nomenclature. The word papilledema should be restricted to the swelling of the optic nerve head due to intracranial pressure, while similar swelling of the nerve due to infections should be designated as optic neuritis with edema. While the edema in these two classes of cases may look the same when viewed with the ophthalmoscope, they are two entirely distinct conditions etiologically and should be so designated. I was rather surprised to hear Dr. Wheeler say that he did not think infection in teeth and tonsils ever produced these optic nerve disturbances, or words to that effect. I do not see what stronger proof one can give of the etiology in these cases than the almost immediate beneficial effect on the vision by the extraction of an infected tooth or the removal of infected tonsils. The benefits of this procedure on all types of neuritis is everyday knowledge. If the removal of a focus of infection in a tooth or tonsils is followed by the rapid subsidence of the inflammation in a joint or the pain or loss of function of a nerve, what better proof can we have of the cause of the inflammation or loss of function?

One of the cases which I did not read for fear of boring you had an acute retrobulbar neuritis which was attributed to an abscessed tooth. The vision

began to improve within 24 hours after the tooth was extracted. Six months later there was a recurrence of the retrobulbar neuritis, which was again promptly relieved by the extraction of another infected tooth. Some months later extreme vertigo confined this patient to her room for some weeks and was promptly relieved by the extraction of infected impacted third molars. Dental infection hit this patient's optic nerve twice and the vestibular branch of the auditory once. Relief promptly followed the removal of the infecting teeth. It might be well to consider briefly what happens in the body from these foci of infection. While in some instances there is more or less of a constant pouring into the blood of bacteria and toxins, in other cases this is intermittent and only happens when the inflammatory barriers about the focus are ruptured. In these intermittent cases it takes some time for the body to get rid of the infection. While the infection is active we get marked symptoms. Any of the joints or nerves throughout the body may be affected; but as the blood gradually counteracts this infection by its antitoxins the symptoms subside. This shot into the blood stream may last one or two weeks, with its accompanying sequel of blindness, vertigo, neuritis or joint inflammation. When the fighting forces of the body overcome these toxins, the symptoms subside. The pain and swelling in the joints or the loss of function of some special nerve. Undue credit is therefore given to so many of these nasal operations which are undertaken at about the time Nature is winning her own battle. It is so easy to attribute the cure to an unnecessary operation, while as a matter of fact it took place not because of, but in spite of, the operation. Let me confess that I have been as guilty as any of you. Practically all my first 30 or 40 cases were attributed to sinus infection, and I thought, as most of you seem to still think, that there was a direct extension from the infection in the sinuses to the nerve. It was only after I saw that many of these cases make speedy recoveries by simply removing some distant focus, that I began to realize that we must be dealing with hematogenous infection and that direct extension, if it really ever did take place, was rare. In none of the last 30 cases in my series, which now number 123, was there direct extension from the sinuses to the nerve, at least as far as I could determine. Of course in malignant disease of the sphenoid there is direct extension, but these cases are rare.

Chronic retrobulbar neuritis is, I believe, many times due to some constant focus. The removal of infected teeth or tonsils has seemed to prevent further loss of vision in several such cases. After the removal of some definite focus or when no focus can be discovered, nasal treatment directed to the establishment of better ventilation and drainage of the sinuses will, I believe, be found of considerable value. The shrinking of the intranasal structures has in a few cases been followed by some immediately visual relief.

I thank Dr. Douglass for his kind remarks in referring to my change of view. Of course the optic canal still cuts some figure in these cases, but enough has been said about it for the present. The one point I wished to bring to you is the infection from other sources than the sinuses. One is so apt to do some operation and say the operation cured the condition. Let us get away from that. Many of these cases will improve under treatment or even without treatment. One man came to me who could not see anything. I examined his nose and treated it by astringent sprays, hot salin irrigation and hot mineral oil, and before he left the office there was a distinct improvement. I think it is important to treat the nose in these cases to relieve impaired ventilation and drainage.

Thanks for the kind words from Dr. Dwyer, in which he states that bacteria from any focus may hit any nerve in the body, of which the optic is one.